SECTION C: THEORY

GYNECOLOGY

Anatomy

CHAPTER 1

EMBRYOLOGICAL DEVELOPMENT OF FEMALE REPRODUCTIVE TRACT

- Genetic sex is established at fertilization, but the gonads do not begin to attain sexual characteristics until week 7. Early genital development is referred to as the indifferent stage of sexual development and the external genitalia do not acquire distinct masculine or feminine characteristics until week 12.
- Testes and ovaries are derived from the mesodermal epithelium (mesothelium) lining the posterior abdominal wall, the underlying mesenchyme and the primordial germ cells.
- The primordial germ cells form in the wall of the yolk sac during week 4. They later migrate into the developing gonads at week 6 and differentiate into the definitive germ cells (oogonia).
- The reproductive organs in both sexes develop from primordia that are identical at first.
  - Gonads develop at week 5 from thickened mesodermal epithelium on the medial side of the mesonephros, at the gonadal ridge.
  - Primary epithelial sex cords grow into the underlying mesenchyme.
  - During this indifferent stage, an embryo has the potential to develop into either a male or a female. The indifferent gonads consist of a cortex and medulla.
  - In the male (XY) the cortex regresses and the medulla develops. The reverse occurs in the female (XX).
- At first both the male and the female have 2 pairs of genital or sex ducts: the mesonephric (wolffian - medial) and paramesonephric (m llerian - lateral) ducts.
- Gonadal sex is determined by the Y chromosome, which exerts a positive testis-determining action (TDF) on the indifferent gonad.
- In the absence of a Y chromosome and in the presence of 2X chromosomes, ovaries develop, the mesonephric ducts regress, the paramesonephric ducts develop. The superior end of these ducts open into the future peritoneal cavity. The lower end becomes the uterus and uterine tubes
- The vagina develops from the vaginal plate derived from the urogenital sinus, and the indifferent external genitalia develop into the clitoris and labia.

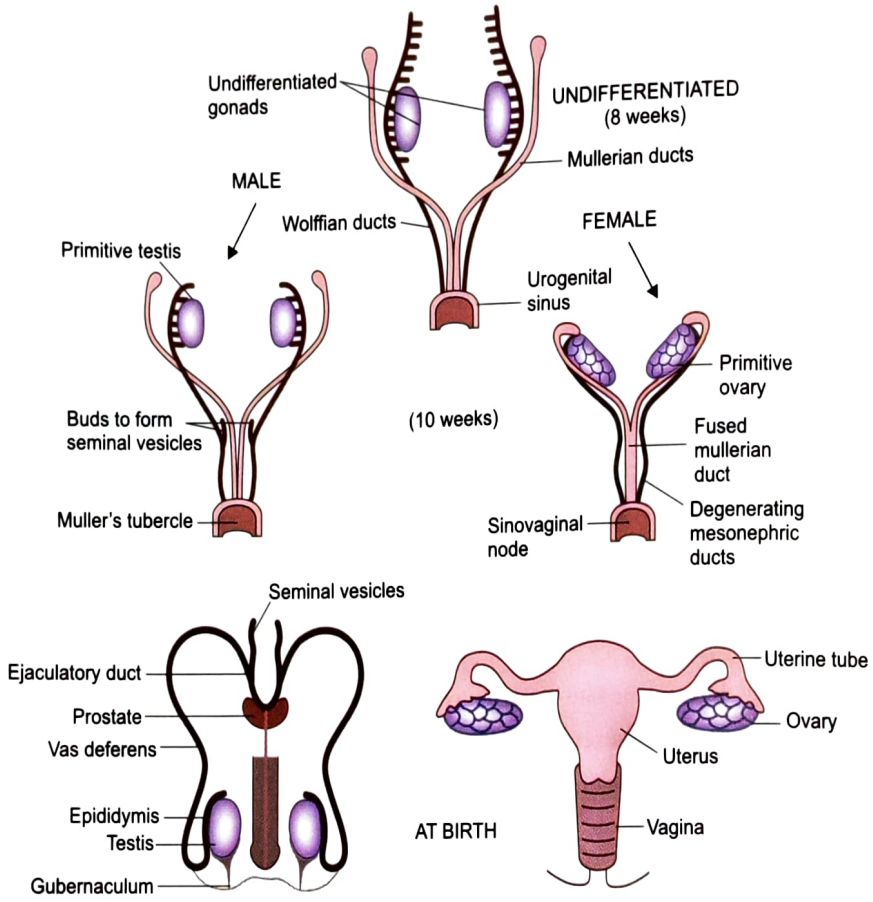


Fig. 1.1: Embroyological development

ANATOMY OF FEMALE REPRODUCTIVE TRACT

Ovaries

- Ovaries are attached to broad ligament by peritoneal fold (mesovarium), attached to uterus by ovarian ligament, and attached to pelvic wall by suspensory ligament.
- 2 major functions – gametogenesis and steroidogenesis.
- Gametogenesis – oogenesis in female.
  - Developing gamete is oocyte and mature gamete is ovum.
- Primary hormones produced are estrogen and progesterone.

- Estrogen promotes growth and maturation of sex organs and promotes formation of female secondary sex characteristics.
- Progesterone prepares uterus for implantation and pregnancy and prepares mammary gland for lactation.

▪ Each ovary is covered with a single layer of squamous or cuboidal cells, possessing microvilli and occasional cilia – germinal epithelium.
- Directly under is tunica albuginea of ovary.
- Underlying tunica albuginea is cortex, which is composed of cellular connective tissue with scattered smooth muscle fibers and ovarian follicles at various stages of development.
- Each ovary has central medulla, which is composed of loose connective tissue and contains blood vessels, lymphatics, nerves.

➢ **Oviducts (Fallopian tubes) –** Divided into infundibulum with fimbriae, ampulla, isthmus, intramural portion.

▪ Infundibulum is funnel-shaped end of oviduct that opens into peritoneal cavity – just prior to ovulation it moves to overlie site of Graafian follicle in order to capture emerging oocyte.

▪ Longest portion is ampulla, which is usually the site of fertilization.
- Function is to nourish both oocyte and sperm and retain them to optimize chances for fertilization.
- Primary means of moving ovum is peristalsis – epithelial cells become shorter and fewer of them are ciliated, mucosa becomes thrown into extremely convoluted folds, muscularis becomes thicker.

▪ Final portions of oviduct function to move fertilized ovum into uterus by peristalsis – isthmus and intramural portion have small lumen, short non-ciliated epithelial cells, few mucosal folds, thick muscularis.

▪ If oviduct doesn't function properly, embryo can implant outside of uterus → ectopic pregnancy → tubal pregnancy in oviduct.

➢ **Uterus –** Provides environment for developing embryo and functions to expel baby at birth.

▪ Divided into body (fundus) and cervix, which extends into vagina.

▪ Wall of uterine body is divided into: myometrium, endometrium, perimetrium.

▪ Myometrium – extremely thick muscle layer that's loosely organized into 3 layers, and serves to protect developing fetus and to expel fetus at birth.
- Size of muscle fibers increases significantly and new muscle cells are produced.
- Undergoes an increase in collagen content during pregnancy.

▪ Endometrium – divided into stratum functionalis and stratum basalis.
- Stratum functionalis proliferates and degenerates during menstrual cycle – composed of simple columnar epithelium with both ciliated and secretory cells.
  * Epithelium invaginates into simple tubular glands – uterine glands.
  * Highly vascularized with spiral arteries that are important during menstruation.
- Stratum basalis – underlies stratum functionalis and is retained during menstruation.
  * Cells act as stem cells to regenerate stratum functionalis with each cycle.
  * Nourished by short straight arteries.
- Endometriosis – endometrial tissue is present in peritoneal cavity and can lead to ectopic pregnancies but can be treated with hormone therapy.

▪ Hormonal control of menstrual cycle – cycle is 28 days.
- Proliferative/follicular stage (day 5-15) – influenced by estrogen from developing ovarian follicles.

- At the beginning of this phase, stratum functionalis is absent and stratum basalis is thin.
- Endometrium rapidly proliferates.
- Epithelial cells from base of glands residing in stratum basalis reconstitute uterine glands and endometrial surface.
- Stromal cells proliferate and spiral arteries regenerate into lower 2/3rd of stratum functionalis.

▪ Secondary/luteal phase (day 16-28) – influenced mainly by progesterone from corpus luteum.
- Uterine glands enlarge and become corkscrew-shaped and begin to secrete glycogen-rich fluid.
- Spiral arteries lengthen, become more coiled, extend nearly to surface

▪ Menstrual phase (day 1-4) – influenced by decline in estrogens and progesterone as corpus luteum degenerates.
- Spiral arteries begin to contract as progesterone levels decline, causing stratum functionalis to become ischemic.
- Surface epithelium disrupted and blood vessels burst.
- Blood, uterine fluid, and sloughing epithelial and stromal cells from stratum functionalis constitute menstrual flow.

➢ **Cervix** – Lower 1/3rd of uterus and does not contain spiral arteries and its epithelium is not sloughed off during menstruation.
▪ Function is to provide tight muscular opening to uterus that allows sperm to enter but provides barrier to infection.
▪ 2 surfaces – endocervix bordering cervical canal and ectocervix, which has its surface facing vaginal lumen.
▪ Endocervix – mucosa is continuous with endometrium and is composed of simple, mucus-secreting columnar cells.
- Thrown into deep crypts – plica palmatae.
- Mucus produced by epithelial cells help to protect against bacterial invasion.

▪ Ectocervix – external surface of part of cervix that projects into vagina.
- Its mucosa is continuous with vaginal lining and has epithelium of stratified squamous cells.
- Abrupt squamocolumnar junction between endocervical and ectocervical epithelia typically occurs at external os.

➢ **Vagina** – Connects uterus to outside of body and acts in transport of sperm to uterus and in expulsion of newborn.
▪ Nonkeratinized stratified squamous epithelium – no glands and no mucus secretion.
▪ Lamina propria has many thin walled blood vessels that contribute to diffusion of vaginal fluid across epithelium.
▪ Mucosa throw into transverse folds/rugae.
▪ Vaginal epithelium responds to hormonal changes during menstrual cycle – glycogen stores in epithelial cells and reaches maximal levels at ovulation, after which time glycogen-rich superficial layer of epithelium is shed.
▪ Breakdown of glycogen by bacteria in vagina produces lactic acid, causing environment to have acidic pH – inhibits growth of other bacteria, bacterial pathogens, fungus, limits the time sperm can survive in vagina.

➢ **Vulva** – Labia majora, labia minora, clitoris, vestibule of vagina.
▪ Labia majora – paired outer folds of relatively adipose epidermal tissue.
▪ Labia minora – hairless inner epidermal folds.

- Clitoris – body of erectile tissue, homologous to corpus cavernosum of penis, which becomes engorged during sexual stimulation.
- Vestibule of vagina contains numerous minor glands.
- Bartholin's glands – paired mucous glands which open to either side of vaginal orifice and homologous to bulbourethral glands of male in that they secrete during sexual arousal.
- Paraurethral glands – form network of tissue in space anterior to vaginal wall, surrounding and opening to urethra.
- Arise from same embryonic tissue that forms prostate.

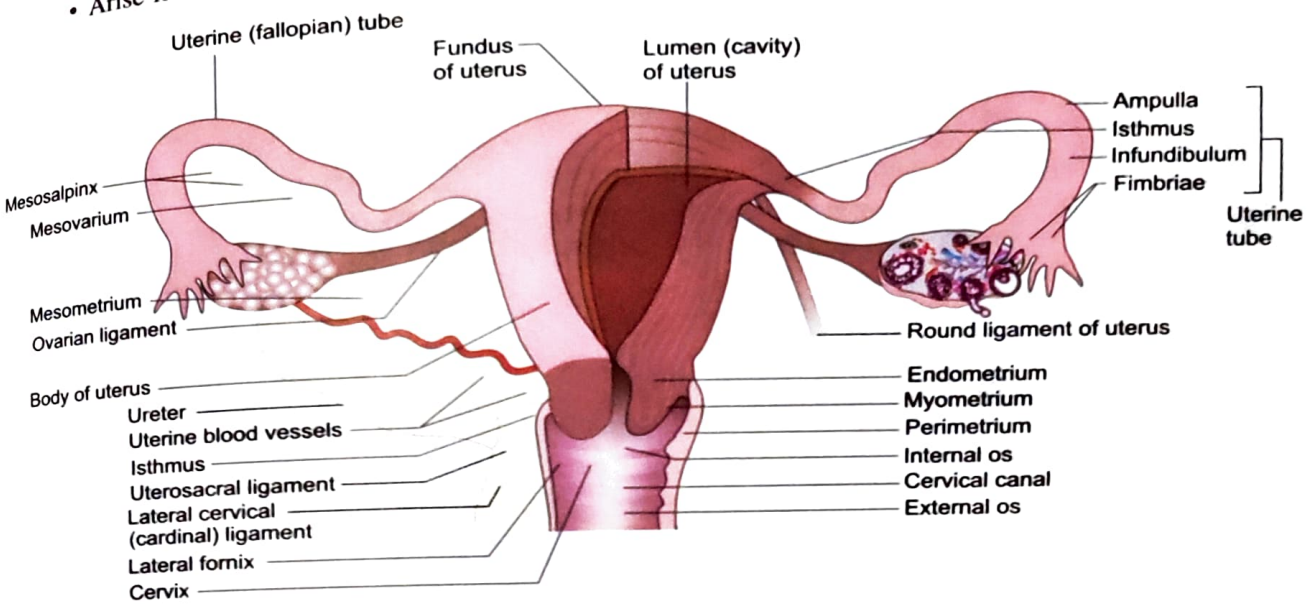


Fig. 1.2: Parts of female reproductive tract

POUCH OF DOUGLAS

- Peritoneal cul-de-sac is situated in the recto vaginal space
- Continuous with the pararectal fossa of either side
- Boundaries
  1. Anterior – peritoneum covering posterior vaginal wall
  2. Posterior – peritoneum covering sigmoid colon and rectum
  3. Lateral – uterosacral ligaments
  4. Floor – reflection of the peritoneum of the peritoneal cavity
- Contents
  1. May remain empty
  2. May contain coils of intestine or omentum
- Surgical importance
  1. Pelvic hematocele or pelvic abscess, because it is the most dependent part

2. Enterocele due to herniation through posterior fornix
3. Vaginal ligation done through this opening in vaginal tubectomy
4. Culdoscopy, culdocentesis and pneumoperitoneum can be done through the pouch
5. Nodules in the pouch may be due to:
   a. Pelvic malignancy
   b. Endometriosis
   c. Genital tuberculosis.

➢ Aspiration of pouch of Douglas

1. Diagnostic purposes:
   a. Pelvic abscess
   b. Ectopic pregnancy in hematocele
   c. To detect malignancy in ascites with ovarian cyst
2. Therapeutic purpose – drainage of pus in pelvic abscess

TRANSITIONAL (TRANSFORMATION) ZONE

- 1-10 mm width, squamo-columnar junction (SCS) between squamous epithelium of vagina and columnar epithelium of endocervix.
- Situated well outside external os but indrawn post menopause.
- Consists of endocervical stroma and glands covered by squamous epithelium
- At birth and during premenarchal years, the SCJ is located at or very close to the external os (original SCJ). During reproductive age, the SCJ is located at variable distances from the external os. In a postmenopausal woman, the new SCJ is not visible and has receded into the endocervix.
- Constantly irritated by hormones, infection and trauma.
- More chances of severe dysplasia, carcinoma-in-situ and even invasive carcinoma.

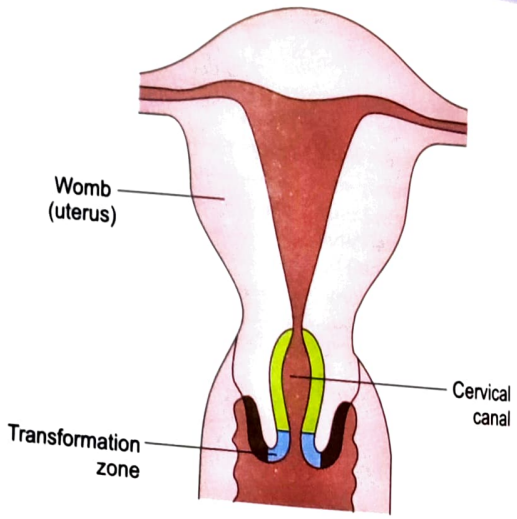


Fig. 1.3: Cross section of uterus showing transformation zone

PELVIC MUSCULATURE

➢ The pelvic muscles of importance in gynecology are those of the pelvic floor. These muscles are grouped into 3 layers:

1. Pelvic diaphragm
2. Urogenital diaphragm
3. Superficial muscles of the pelvic floor.

➢ The levator ani and coccygeus muscles on either sides form the pelvic diaphragm separating the structures in the pelvis from the perineum and ischiorectal fossa.

➢ Levator ani is a wide, thin curved sheet of muscle arising anteriorly from the pelvic surface of the body of the

pubis, the ischial spine and the tendinous arch of the obturator fascia. It has three parts:

1. Puborectalis – the most medial part, encircles and supports the rectum and vagina
2. Pubococcygeus – the strongest part extending from the pubis to the coccyx
3. Iliococcygeus – the most posterior and is also attached to the coccyx.

- Perineal body is a fibromuscular mass occupying the area between the vagina and the anal canal. It supports the lower part of vagina.
- The ligaments in the pelvis are condensations of the pelvic cellular tissue (except the broad ligament). The cardinal and uterosacral ligaments are the strongest supports of the uterus and are called the true supports, while the round ligaments, broad ligaments and ovarian ligaments are called false supports.
- Genital prolapse, urinary stress incontinence and fecal incontinence are all related to laxity and atonicity of the muscles of the pelvic floor, as well as denervation of pelvic nerves during childbirth.

PELVIC LYMPHATICS

1. Inguinal glands
   a. Superficial group
      Horizontal – parallel to inguinal ligament
      Vertical – along the long saphenous vein
   b. Deep inguinal or femoral group
      5-6 in number
      Uppermost gland is called gland of Cloquet or gland of Rosenmuller
2. Parametrial glands
3. Hypogastric glands
   Below bifurcation of common iliac artery
   Also called bifurcation nodes
4. External iliac glands
   a. Lateral – lateral to external iliac artery
   b. Middle – between artery and vein
   c. Medial – medial to vein
   d. In carcinoma cervix, middle and medial groups are involved
5. Sacral group
   a. Lateral group
   b. Medial group
6. Lumbar group
   a. Superior group
   b. Inferior group.

- **Lymphatic drainage of female reproductive system:**
  - The ovaries and fallopian tubes drainage systems follow the infundibulopelvic ligament to the upper para-aortic nodes near the origin of the ovarian arteries.
  - The fundus of the uterus drains via the round ligament to the superficial inguinal lymph nodes and along the ovarian ligament to the para-aortic group of lymph nodes.

- Body of the uterus drains to the external iliac lymph nodes.
- Cervix drains to the external and internal iliac, obturator and sacral nodes.

  (Note: The lymphatic drainage of cervix is through lateral, anterior and posterior channels. Usually the spread of cancer to para-aortic nodes is progressive, but occasionally it may metastasize directly to the para-aortic nodes by embolization through the posterior channels)
- The lower vagina drains to the inguinal and femoral nodes in the same way as the vulva. The drainage of the middle and upper vagina is the same as that of the cervix.
- The vulva mainly drains into the superficial inguinal lymph nodes which then drain into the deep inguinal and femoral lymph nodes.

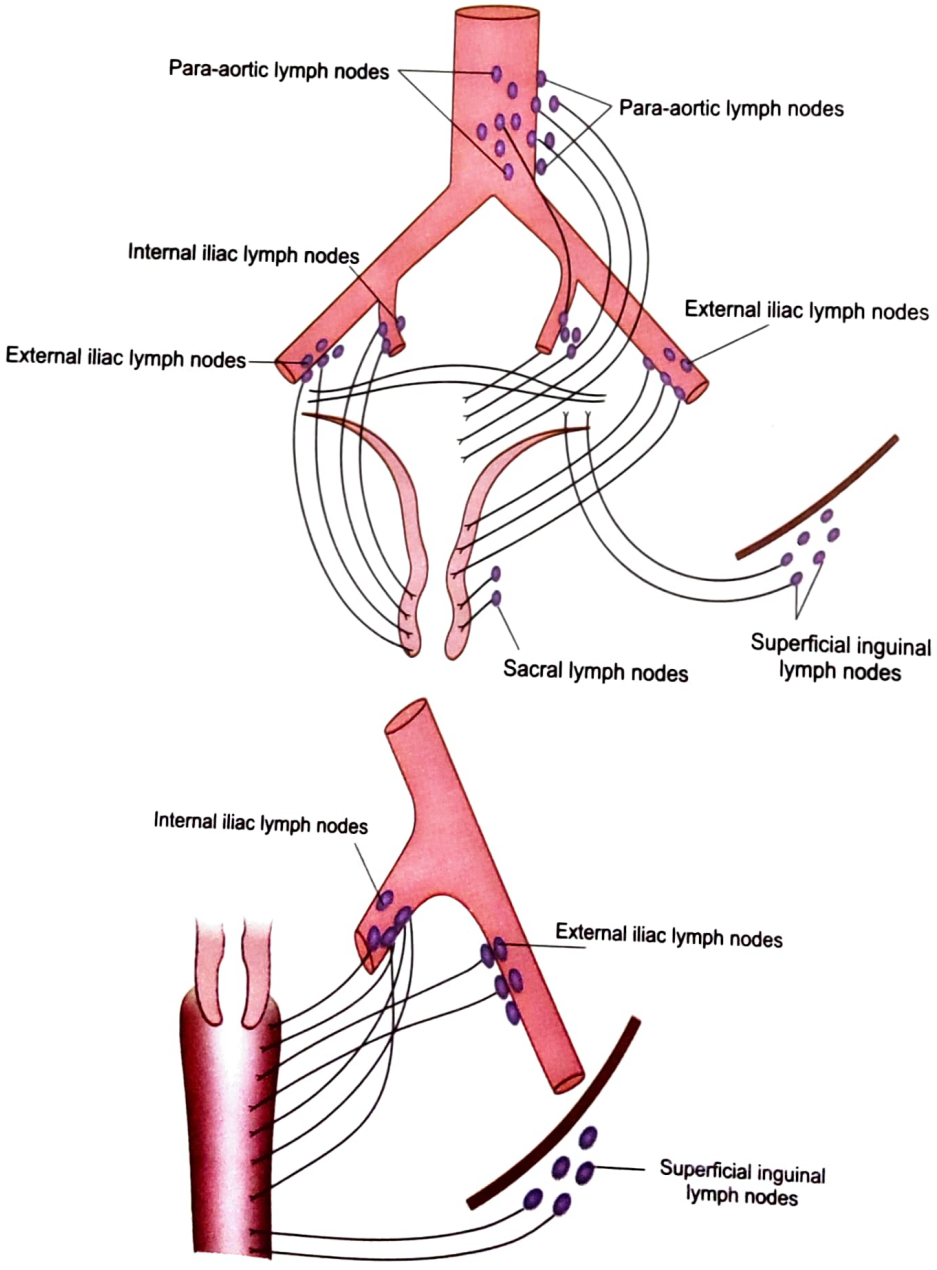


Fig. 1.4: Lymphatic drainage

PREVIOUS EXAMINATION QUESTIONS	
Long Essays	1. Write the anatomy and functions of pelvic diaphragm.
Short Essays	1. Course of ureter in pelvis. 2. Components of pelvic floor. 3. Muscular supports of uterus. 4. Describe supports of uterus. 5. Describe the lymphatic drainage of cervix and its clinical significance. 6. Pouch of Douglas. 7. Levator ani. 8. Anatomy of fallopian tube. 9. Muscles of pelvic floor. 10. Pelvic cellular tissue. 11. Pelvic floor. 12. Vascular supply of ovary. 13. Vascular supply of uterus. 14. Fallopian tube.
Short Answers	1. Embryological development of the uterus. 2. Clinical significance of pouch of Douglas. 3. Anatomy of fallopian tube with figure. 4. Perineal body. 5. Blood supply to uterus. 6. Lymphatic drainage of vulva. 7. Importance of transformation zone of cervix. 8. Embryological remnants of Wolffian system.

Normal Histology

CHAPTER 2

FOLLICULOGENESIS

- Primordial follicle – single layer of flattened follicular cells surround small primary oocyte.
  - Oocyte typically has large round euchromatic nucleus with prominent dark nucleolus.
  - Inactive ovarian follicles – tend to be located in superficial cortex beneath dense tunica albuginea.
- Primary follicle – oocyte has grown in size and surrounded by cuboidal to columnar granulosa cells.
  - Zona pellucida – glucosaminoglycan rich zone forms between granulosa cells and oocyte.
  - Theca folliculi – developing in area immediately surrounding stroma, with external fibrous layer (theca externa) and internal steroidogenic layer (theca interna) that is well vascularized.
  - Theca interna contains clusters of foamy-appearing theca interna cells – secrete estrogen precursors that cross granulosa cells of follicle that complete synthesis of estrogens.
- Secondary follicle – marked by formation of centrally located antrum that appears between granulosa cells.
  - Membrana granulose can now be separated into granulose proper, culmulus oophorus, corona radiata.
- Graafian follicle – very large follicle just before ovulation.
- Atretic follicle – granulosa cells undergo degeneration by apoptosis, and their distinctly small dense pyknotic nuclei appear among normal granulosa cell nuclei.
  - Remnants of larger ovarian follicles may appear as wavy collagenous scars but most atretic follicles are completely phagocytosed by macrophages.
  - May be seen at any stage of development.
- Corpus luteum – forms following ovulation and expulsion of oocyte.
  - Walls of empty follicle collapse and fold extensively.
  - Following ovulation, granulosa cells enlarge and become secretory – basement membrane surrounding follicle breaks down and allows small blood vessels to penetrate previously avascular layer of granulosa cells.
  - Temporary endocrine gland – formed from remnants of Graafian follicle's granulosa cells and theca interna cells.

- Granulosa lutein cells (progesterone) are large pale spherical whereas theca lutein cells (estrogen) are smaller, have less cytoplasm and often stain poorly – both cells secrete steroids.

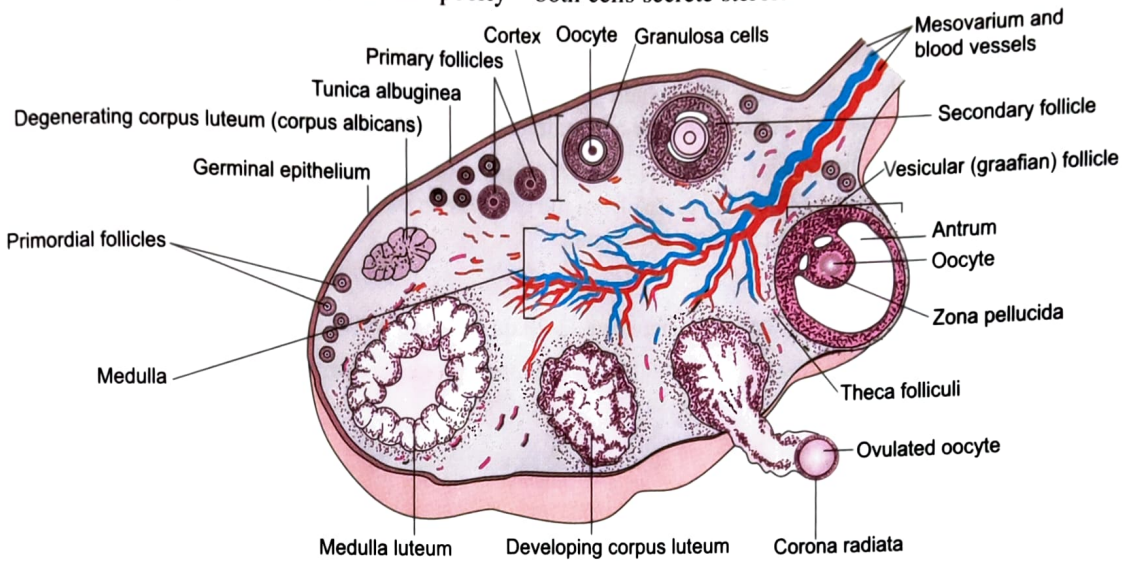


Fig. 2.1: Folliculogenesis

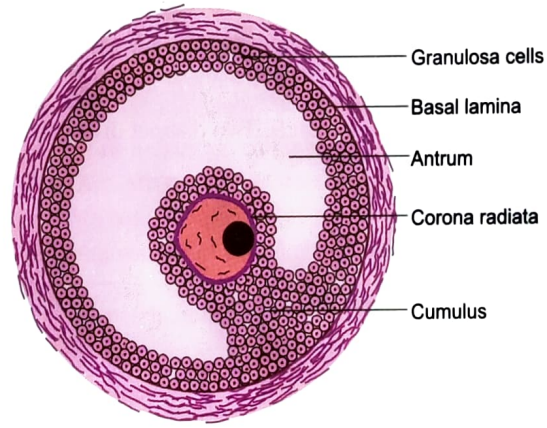


Fig. 2.2: Graffian follicle

CORPUS LUTEUM

- After ovulation, the ruptured graffian follicle develops into corpus luteum.
- **Stages of the life cycle**
  1. Proliferation
  2. Vascularization
  3. Maturation
  4. Regression.
- **Hormones** in relation to corpus luteum:

1. FSH in presence of high level of estrogen induces LH receptors in the granulosa cells of the dominant follicle. Thereafter, mid-cycle LH surge causes luteinization of the granulosa cells and progesterone secretion. LH secretion must be continued for the function of corpus luteum, failing which the corpus luteum will regress.
2. Adequate folliculogenesis in the preovulatory phase with increased secretion of estradiol and of 17 alpha-hydroxy progesterone is a prerequisite for adequate corpus luteum formation.
3. Corpus luteum has a lifespan of about 12-14 days. The cause of degeneration in an infertile cycle is unknown. It has been suggested that $PGF_{2\alpha}$ liberated from the ovary is luteolytic. Estradiol is also considered to have luteolytic effect. The role of prolactin is unclear.

➢ **Corpus luteum** of pregnancy

1. There is hyperplasia of all the layers between 23-28 days due to hCG
2. hCG, like LH, will stimulate the corpus luteum to secrete progesterone
3. K cells are also increased in number
4. Growth reaches its peak at about 8^{th} week when it measures about 2-3 cm
5. It looks bright orange, later on becomes yellow and finally pale
6. Regression occurs following low levels of hCG
7. Degenerative changes take place most frequently at about 6 months of gestation

ENDOMETRIUM OF THE UTERUS

➢ The endometrium is the lining epithelium of the uterine cavity above the level of internal os. It consists of surface epithelium, glands, stroma and blood vessels. Two distinct divisions are established:

1. Basal zone (stratum basalis)
2. Functional zone.

➢ **Basal Zone** (deeper layer)

1. It is about $1/3^{rd}$ of the total depth of the endometrium.
2. It measures about 1 mm.
3. It lies in contact with the myometrium.
4. It is uninfluenced by hormones and therefore, no cyclic changes are observed.
5. After shedding of the superficial part during menstruation, the regeneration of all the components occurs from this zone.

➢ **Functional Zone** (superficial layer)

1. This zone is under the influence of fluctuating cyclic ovarian hormones, estrogen and progesterone.
2. The changes in different components during an ovulatory cycle has been traditionally divided into 3 stages:
   a. Proliferative phase - extends from 5^{th} or 6^{th} day to 14^{th} day (till ovulation)
   b. Secretory phase - begins on day 15 and ceases 5-6 days prior to menstruation
   c. Menstruating phase - degeneration and casting off of an endometrium prepared for a pregnancy.

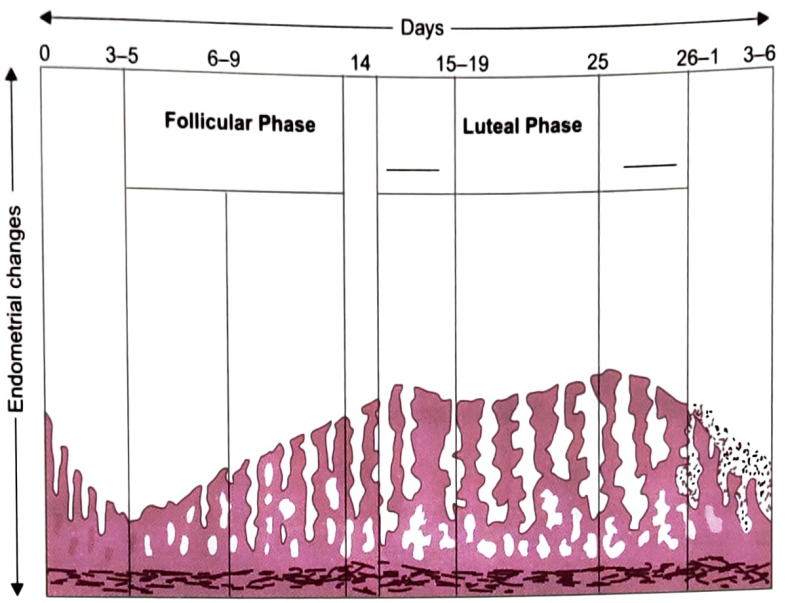


Fig. 2.3: Change in endometrium with each menstrual cycle

PREVIOUS EXAMINATION QUESTIONS	
Long Essays	1. Draw a diagram and label corpus luteum. 2. Draw diagram of a Graafian follicle. 3. Microscopic picture of secretory endometrium. 4. Microscopy of proliferative endometrium.
Short Answers	1. Corpus luteum

Physiology

CHAPTER 3

MENSTRUAL CYCLE

➢ Phases of menstrual cycle

- The *menstrual phase* begins on day 1 with the onset of bleeding and continues for several more days until the shedding of the endometrial lining stops (usually day 4–5).
- The *proliferative phase* begins at the end of the menstrual phase and ends at ovulation (usually day 13 or 14). This phase is characterized by endometrial thickening and ovarian follicular maturation.
- The *luteal (secretory) phase* starts at ovulation and lasts through day 28 before the entire process starts over again on day 1.

➢ Biologic basis of menstruation

Coordination of the menstrual cycle depends on a complex interaction of the brain, pituitary, ovaries, and endometrium.

a. Brain

- The hypothalamus is located at the base of the brain and essentially functions as the central processing unit of the reproductive system.
- Neuronal stimuli from the cerebral cortex are converted by the hypothalamus into pulses of neuropeptides (gonadotropin-releasing hormone, GnRH).
- Hypothalamic GnRH production is modulated by negative feedback of steroid hormones (estradiol-17 , progesterone).

b. Pituitary

- Located just below the hypothalamus, the pituitary gland consists of the neurohypophysis (posterior lobe) and the adenohypophysis (anterior lobe). It is the anterior pituitary that gives rise to gonadotropin (luteinizing hormone [LH] and follicle-stimulating hormone [FSH]) production.
- Pulsatile GnRH from the hypothalamus initiates the synthesis and secretion of LH and FSH. Similar to the hypothalamus, the anterior pituitary is subject to negative feedback regulation by the steroid hormones.
- In women of reproductive age, LH and FSH levels generally remain in the 10–20 mIU/mL range. After the menopause or surgical oophorectomy, estradiol-17 levels decline and LH and FSH are released from negative feedback, achieving circulating concentrations of more than 50 mIU/mL.

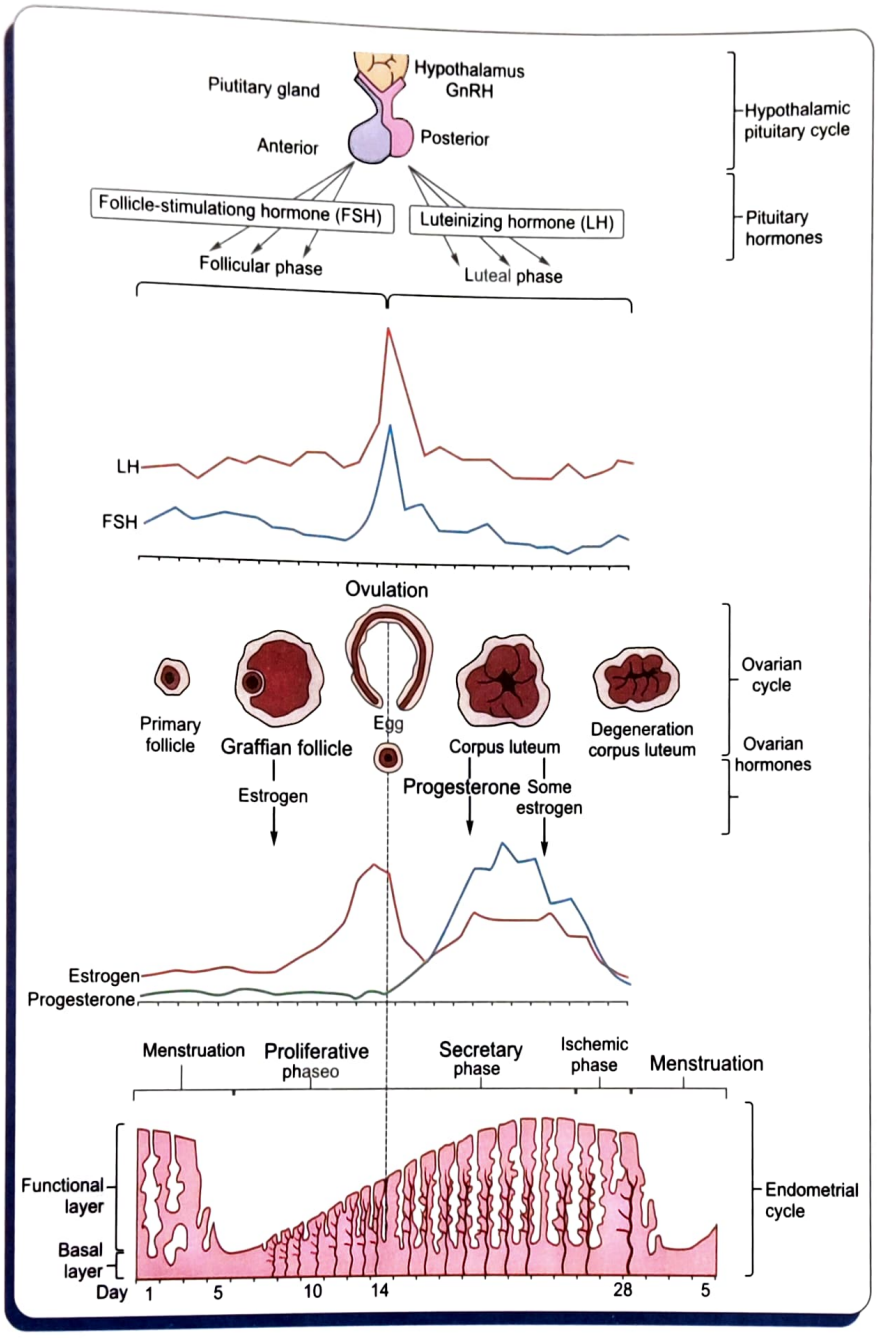


Fig. 3.1: Physiology of menstrual cycle

c. Ovaries

- Primitive germ cells (oogonia) divide by mitosis during fetal embryogenesis, peaking at around 7 million by 5 months of gestation.
- Meiotic division then begins, resulting in formation of primary oocytes. However, rapid atresia reduces the number of available follicles to 2 million at birth. At puberty, only around 300,000–400,000 follicles remain.
- Oocytes remain "resting" in meiotic prophase until puberty. Resting ovarian follicles are surrounded by thecal and granulosa cells. FSH stimulates the granulosa cells and LH stimulates the thecal cells.
- Only a single "dominant follicle" develops each menstrual cycle. When it produces enough estrogen to sustain a circulating estradiol-17 concentration of about 200 pg/mL for 48 hours, the hypothalamic–pituitary axis responds by secreting a surge of gonadotropins, primarily LH. This LH surge precedes ovulation by 24–36 hours.
- Following ovulation, the follicle collapses to form the corpus luteum. This endocrine organ mainly synthesizes progesterone to prepare the endometrium for pregnancy.
- If implantation does not occur, the corpus luteum will degenerate, resulting in a precipitous decline in circulating steroid hormone levels and the onset of menstruation. The decreasing steroid hormone levels release the negative feedback mechanism, inducing the pituitary to increase gonadotropin secretion. As a result, a new cycle of follicular recruitment is initiated.
- If implantation does occur, the embryo will rescue the corpus luteum by producing human chorionic gonadotropin (hCG) to prevent menstruation. At 7–9 weeks of gestation, the placenta takes over the production of progesterone from the corpus luteum.

d. Endometrium

- Monthly cyclic changes occur in the endometrium under the control of steroid hormones produced by the ovaries.
- Estradiol-17 production by the ovarian follicles induces endometrial proliferation. Progesterone synthesis by the corpus luteum then acts to mature the estrogen-primed endometrium in preparation for blastocyst implantation.
- Lowered steroid hormone levels in the late secretory phase cause a collapse of the endometrial vasculature, resulting in menstruation.

Mittelschmerz: Mittelschmerz pain is one-sided, lower abdominal pain in women. It occurs at or around the time of ovulation. It is seen in 20% of women. No treatment is needed usually. Pain killers may be taken for severe pain.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. Explain physiology of ovulation.
Short Essays	1. Phases of menstrual cycle. 2. Explain physiology of ovulation. 3. Progestogens and their applications in gynecology. 4. Menstruation.
Short Answers	1. Enumerate hormones secreted by pituitary gland. 2. Ovulation. 3. Menstruation. 4. Mittlelschmerz.

Pediatric Gynecology, Adolescent Problems and Puberty

CHAPTER 4

PUBERTY

➢ Definition

Puberty is the period of transition from childhood to adulthood. Involves physical growth, development of sex organs and secondary sexual characters and attainment of menarche culminating in complete physical and psychological maturation. This period usually takes 8 to 18 yrs.

➢ Timing of puberty

The major factor that determines the timing of puberty is genetic. The other factors like geographic location, exposure to light, general health and nutrition and psychological factors influence the time of initiation and rate of progression.

➢ Pubertal events

The events occur in the following sequence.

1. Physical growth

   Growth spurt occurs at about 11-12 years, and doubles in one year. The increase in height is between 6 and 11 cm. The growth peak is attained 1 year prior to menarche. This is due to increased levels of oestrogen and growth factor I.

2. Changes in genital organs
   - Ovaries show follicular enlargement. There is stromal cell hyperplasia and the ovaries become bulky and oval in shape.
   - Uterus shows endometrial and myometrial proliferation. The ratio of uterine body to the cervix changes from 1:2 at birth, to 1:1 at menarche.
   - Vaginal epithelium becomes stratified squamous non-keratinized, and acquires glycogen due to the effect of estrogen. It becomes host to a profusion of Doderlein's bacilli that convert glycogen to lactic acid. Thus the vaginal pH changes from alkaline in the prepubertal period to acidic (4-5) in the reproductive period.
   - The vulva becomes more reactive to steroid hormones.
   - Mons pubis and labia minora also increase in size.
3. Secondary sexual characters
   - Breast – There is ductal proliferation and fat deposition in response to oestrogens.

- Pubic and axillary hair – increased secretion of adrenal sex steroids from 7 yrs of age is mainly responsible for increased sebum formation, pubic and axillary hair and change in voice.
- Female body habitus – estrogens lead to subcutaneous fat deposition, leading to the typical female body habitus.

4. Menarche is the first menstruation in life.
   - It usually occurs between 9 to 17 yrs. It is chiefly controlled by genetic factors.
   - It is the final endocrine hallmark of puberty and also denotes an intact hypothalamo-pituitary axis, functioning ovaries, endometrium responsive to endogenous ovarian steroids and a patent uterovaginal canal.
   - In the earlier stages the menstrual cycles are anovular, but after two to three years they regularize and become ovular.

➢ Sequence of pubertal events

Adrenarche → increase in gonadal estrogen (gonadarche) → rapid increase in skeletal growth resulting in a growth spurt → breast development, female fat distribution, vaginal and uterine growth (thelarche) → menarche.

➢ Management of puberty

Puberty involves radical changes that occur at such a pace that an adolescent mind finds them difficult to comprehend and deal with.

1. Nutrition – Since this is the period of rapid growth, should be given foods rich in proteins. Anemia is a common problem and periodic deworming could be carried out.
2. Personal hygiene – Menarche involves the ability to deal with monthly cyclical bleeding. Proper use and disposal of sanitary napkins and regular baths.
3. Sex education.

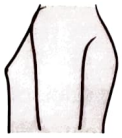
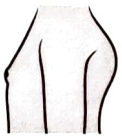
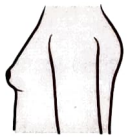
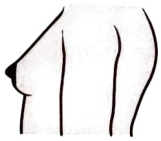
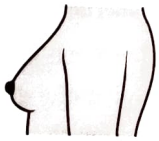
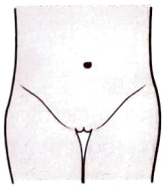
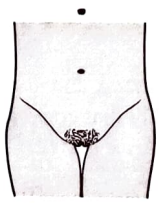
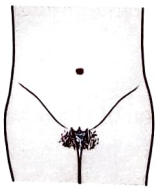
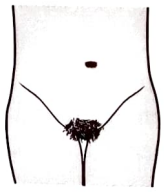
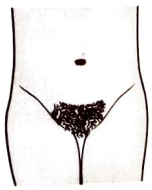
	1	2	3	4	5
Breast	 Prepubertal	 Breast and papilla are elevated as a small mound. Areolar diameter increases	 Further enlargement of the breast bud with loss of the contour separation between breast and areola	 Areola and papilla form a secondary mound	 Mature areola is part of the general breast contour
Pubic hair	 Prepubertal	 Space lightly pigmented chieny along the medial border of the labia majora	 Darker beginning to curl, increased amount spreading over the mons	 Increased amount of coarse, curly but limited to the mons	 Adult feminine triangle with spread to the medial surface of the thighs

Fig. 4.1: Development of breast and pubic hair (Tower stages)

➢ **Tanner Stages of Breast Development**

Stage 1 – Preadolescent: Elevation of papilla only

Stage 2 – Breast bud stage: Elevation of breast and papilla, areolar enlargement

Stage 3 – Further enlargement of breast and areola without separation of contours

Stage 4 – Projection of areola and papilla to form a secondary mound

Stage 5 – Mature stage: Projection of papilla only as areola recesses to breast contour.

➢ **Tanner Stages of Pubic hair Development**

Stage 1 – Prepubertal (can see velus hair similar to abdominal wall)

Stage 2 – Sparse growth of long, slightly pigmented hair, straight or curled, at base of penis or along labia

Stage 3 – Darker, coarser and more curled hair, spreading sparsely over junction of pubes

Stage 4 – Hair adult in type, but covering smaller area than in adult; no spread to medial surface of thighs

Stage 5 – Adult in type and quantity, with triangular distribution ("feminine").

PRECOCIOUS PUBERTY

➢ **Definition**

1. Secondary sexual characters appear before the age of 8 years
2. Menarche before 10 years of chronological age.

➢ **Causes**

1. GnRH dependent (80%) – complete, central, isosexual or true
   a. Constitutional or idiopathic (commonest)
   b. Juvenile primary hypothyroidism
   c. Intracranial lesions
      1) Trauma
      2) Tumor – craniopharyngioma, neurofibroma
      3) Infection – meningitis, encephalitis
2. GnRH dependent – incomplete
   a. Premature thelarche – isolated development of breast tissue before 8 years
   b. Premature pubarche – isolated development of axillary and/or pubic hair prior to the age of 8 years without other signs of precocious puberty
   c. Premature menarche – isolated event of cyclic vaginal bleeding without any other signs of secondary sexual development
3. GnRH independent – precocious pseudopuberty or peripheral – due to excess estrogen or androgen
   a. Ovary
      1) Granulosa cell tumor
      2) Theca cell tumor
      3) Leydig cell tumor
      4) Chorionic epithelioma
      5) Androblastoma
      6) McCune-Albright syndrome

b. Adrenal
   1) Hyperplasia
   2) Tumor
c. Liver – hepatoblastoma
d. Iatrogenic – estrogen or androgen intake.

➢ **Treatment**

Suppression of menstruation using GnRH analogues, i.e., GnRH 100 g intranasal BD – 6 months.

DELAYED PUBERTY

➢ **Definition**

1. Secondary sexual characters do not appear by the age of 14 years
2. Menarche is not established by 16 years of age.

➢ **Causes**

1. Hypergonadotrophic hypogonadism
   a. Gonadal dysgenesis – 46 XO
   b. Pure gonadal dysgenesis – 46 XX, 46 XY
   c. Ovarian failure – 46 XX
2. Hypogonadotrophic hypogonadism
   a. Constitutional delay
   b. Chronic illness
   c. Malnutrition
   d. Primary hypothyroidism
   e. Kallmann's syndrome (isolated gonadotrophin deficiency)
   f. Intracranial tumors – craniopharyngioma, pituitary adenomas
3. Anatomical causes
   a. Mullerian dysgenesis
   b. Imperforate hymen
   c. Transverse vaginal septum.

PUBERTY MENORRHAGIA

- Anovulatory cycles with unopposed and excess estrogen causing endometrial hyperplasia and prolonged excess menstruation.
- If untreated may cause anemia.
- Treatment – Progestogens.
- Very rarely, when there is no response to medical treatment, a mirena coil (progesterone releasing IUCD) can be inserted to avoid surgical intervention and high doses of hormones.

LABIAL ADHESION / FUSION

➢ **Definition –** Labial adhesion is a medical condition of the female genital anatomy where the labia minora become fused together.

- **Signs and symptoms:** It is never present at birth, but rather acquired later in infancy, since it is caused by insufficient estrogen exposure and newborns have been exposed to maternal estrogen in utero. It typically presents in infants at least 3 months old. Most presentations are asymptomatic and are discovered by a parent or during routine medical examination. In other cases, patients may present with associated symptoms of dysuria, urinary frequency, refusal to urinate, or post-void dribbling/vaginal discharge due to pooling of urine in the vulval vestibule or vagina.
- **Diagnosis:** The condition can be diagnosed based on inspection of the vulva. In patients with labial adhesion, a flat plane of tissue with a dense central line of tissue is usually seen when the labia majora are retracted, while an anterior opening is usually present below the clitoris.
- **Pathophysiology:** The primary contributing factor is low estrogen levels. Conditions causing irritation, such as infection, inflammation and trauma, cause the edges of the labia minora to fuse together. Most labial adhesions resolve spontaneously before puberty as estrogen levels increase and the vaginal epithelium becomes cornified.
- **Treatment:** Not necessary in asymptomatic cases. The standard method of treatment in symptomatic case is the application of topical estrogen cream onto the areas of adhesion. In severe cases where the labia minora are entirely fused, causing urinary outflow obstruction or vaginal obstruction, the labia should be separated surgically.
- **Complications:** Urinary tract infection, vulvar vestibulitis and inflammation caused by chronic urine exposure. In severe cases, anuria and urinary retention.

PREVIOUS EXAMINATION QUESTIONS	
Long Essays	1. Define puberty. Describe all physiological changes that occur in normal puberty.
Short Essays	1. Approach to the management of puberty menorrhagia. 2. Differential diagnosis of abnormal bleeding in a pubertal girl. 3. Define precocious puberty. What are the common causes? 4. Tanner staging of breast and pubic hair.
Short Answers	1. Labial adhesions in a child – causes and treatment. 2. Precocious puberty. 3. Premature menarche. 5. Tanner staging of breast development. 5. Delayed puberty. 6. Delayed menarche.

Menopause, Premature Menopause and Postmenopausal Bleeding

CHAPTER 5

MENOPAUSE

- **Menopause** – It is the time of cessation of ovarian function resulting in permanent amenorrhea. It takes 12 months of amenorrhea to confirm that menopause has set in. Therefore, it is a retrospective diagnosis.
- **Climacteric** – It is the phase of waning ovarian activity. It may begin 2-3 years before menopause and continue for 2-5 years after it.
- **Age** - Menopause normally occurs between 45-50 years. The average age is 47.
  1. Premature menopause – This is menopause setting in before the age of 40. Smoking and genetic disposition may induce premature menopause.
  2. Late menopause – It is not uncommon to see a woman menstruate beyond the age of 50. This may be related to good nutrition and better health. It is also common in women with uterine fibroids and those at high risk for endometrial cancer.
- **Symptoms**
  1. **Menstrual** – The 3 classical ways in which the menstrual period ceases are:
     a. Sudden cessation
     b. Gradual diminution in the amount of blood loss with each regular period until menstruation stops
     c. Gradual increase in the spacing of the periods until they cease for atleast a period of 1 year.
  2. **Menopausal** – The most common and most noticeable symptoms of hot flushes and sweating are the hallmark of the climacteric in most women.
     a. Hot flushes are the waves of vasodilatation affecting the face and the neck and these last for 2-5 minutes each. These are followed by severe sweating. Several of these flushes occur in a day, but are more severe during the night, and can disturb sleep. The hot flushes are sometimes preceded by headache. With passage of time, the frequency and severity of flushes diminish over a period of 1-2 years.
     b. Palpitation and anginal pains may be felt.
     c. Mental depression due to disturbed sleep or otherwise, irritability and lack of concentration are noticed.
     d. Some women develop a condition of pseudocyesis.

e. Cancer phobia may also develop.

f. The woman also starts worrying over her looks.

3. **Neurological** – Vasomotor symptoms and paraesthesia take the form of sensations of pins and needles in the extremities.
4. **Libido** – Many notice decreased libido after menopause. Sexual feeling and libido may increase in some, if they feel happy to get rid of menstruation and fear of pregnancy.
5. **Urinary** – Dysuria, stress incontinence and urge incontinence, recurrent infection.
6. **Genital** – Dry vagina, dyspareunia.
7. **Locomotor system** – Menopausal arthropathy, osteoarthritis, fibrositis, backache.
8. **Late sequelae**
   a. Arthritis, osteoporosis, fracture
   b. Cardiovascular accidents – IHD, MI, atherosclerosis
   c. Stroke
   d. Alzheimer disease
   e. Anocolonic cancer
   f. Skin changes
   g. Tooth decay.

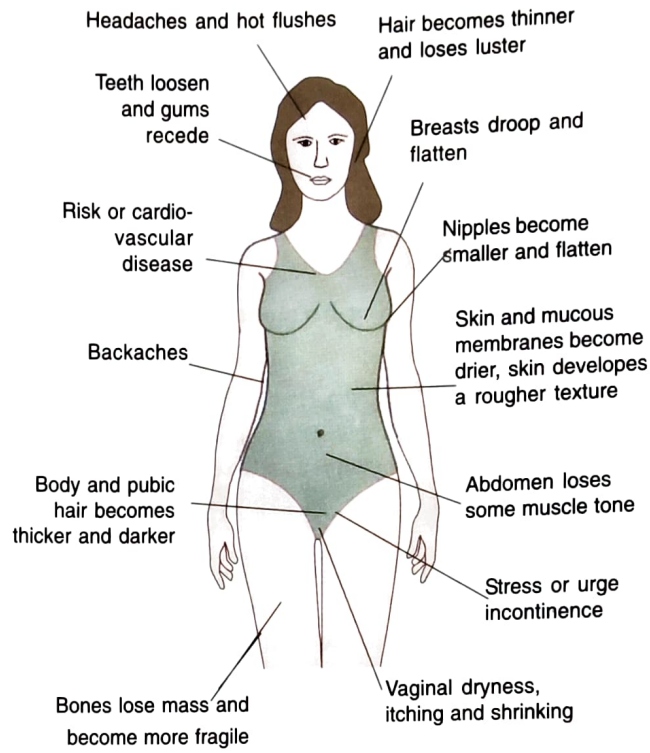

Headaches and hot flushes
Hair becomes thinner and loses luster
Teeth loosen and gums recede
Breasts droop and flatten
Risk or cardio-vascular disease
Nipples become smaller and flatten
Skin and mucous membranes become drier, skin developes a rougher texture
Backaches
Body and pubic hair becomes thicker and darker
Abdomen loses some muscle tone
Stress or urge incontinence
Bones lose mass and become more fragile
Vaginal dryness, itching and shrinking

Fig. 5.1: Postmenopausal problems

➢ **Anatomical changes**

1. Genital organs undergo atrophy and regression
2. Cervix becomes smaller and the fornices gradually disappear
3. Vulva disappears and vaginal orifice narrows down
4. Pubic hair decreases and becomes grey
5. Loss of tone of ligaments supporting uterus and vagina
6. Fat deposition around the breast, hip and abdomen
7. Estrogen deficiency leading on to arthritis and osteoporosis.

➢ **Investigations**

1. History of various symptoms
2. General examination – BP, breasts, weight, hirsutism
3. Pelvic examination – Pap smear

4. Blood sugar and lipid profile
5. Mammography
6. Pelvic ultrasound
7. Bone density study
8. Vaginal cytology – shows features of low estrogen
9. Serum estrogen level (<20 pg/ml) and serum FSH and LH level (>30 IU/l) - to decide on need of HRT.

➢ **Management**

1. Counselling/Non-hormonal treatment

a. The woman often develops pregnancy and cancer phobia. It is the duty of the gynecologist to convince her, after thorough examination and investigations, that all is well with her.

b. Advice on contraceptives is needed. Until menopause is well-established and amenorrhea has lasted for 12 months, couple is advised to use barrier method.

c. Diet should include at least 1.2 g of calcium, vitamin A, C and E. Soya beans are good.

d. Weight bearing exercises prevent or delay osteoporosis.

e. Yoga, meditation and social work can reduce mental stress.

f. Clonidine may be used to reduce the severity and duration of hot flushes.

2. Mild Tranquilizers – These relieve the woman's anxiety, sleeplessness and depression. Anti-depressant like sulpiride may be needed.

3. Hormone Replacement Therapy (HRT)

a. *Indications* – HRT is indicated in women who are:

1) Symptomatic – Women with the aforementioned symptoms need to be treated with HRT on a short-term basis for a period of 3-6 months.

2) High risk cases for cardiovascular disease, osteoporosis, Alzheimer disease, and colonic cancer. These women may benefit from the cardioprotective effect of prophylactic estrogen. Estrogen deficiency increases the risk of atherosclerosis, IHD and angina in a postmenopausal woman. Estrogen is therefore cardioprotective. It also increases HDL and decreases LDL, cholesterol and triglycerides. Estrogen is most effective when taken orally. Estrogen and tibolone are strongly cardioprotective.

3) Those who demand HRT as prophylaxis – HRT may delay or prevent the occurrence of Alzheimer disease.

b. Drugs used: HRT includes estrogen alone or in combination with progesterone. If the uterus is present, progestogens are prescribed in addition to prevent endometrial hyperplasia and further carcinoma.

- Estrogens – Oral (continous combined/cyclic)
  Vaginal cream
  Percutaneous cream
  Subdermal implants
  Transdermal patches
- Tibolone – synthetic compound with a mixed estrogenic,progestogenic and androgenic action. Useful for vasomotor, psychological, libid problems and osteoporosis prevention.

- Bisphosphonates – Etidronate, Alendronate, Risedronate inhibit bone resorption and have protective effect on osteoporosis.
- Selective Estrogen Receptor Modulators (SERMs) – Have estrogenic effect on bone and lipids and antagonistic effect on the breast. Not suitable for vasomotor symptoms. Eg. Raloxifene.

c. *Contraindications*

1) Breast cancer, uterine cancer or family history of cancer
2) Previous history of thromboembolism
3) Liver and gall bladder disease

➢ **Artificial menopause: Can occur following surgery or radiation.**

Sugical: Menstruating women with bilateral oophorectomy and/or hysterectomy

Radiation: Ovarian function is suppressed by external gamma radiation in women <40 yr old. The castration is not permanent. Menstruation may resume after 2 years and even conception is possible.

POSTMENOPAUSAL BLEEDING

➢ **Definition** – Vaginal bleeding occurring anytime after 6 months of amenorrhea in a menopausal age

➢ **Etiology**

1. Vulva – vulvitis, benign and malignant lesions, trauma
2. Vagina – senile vaginitis, benign and malignant vaginal tumor, foreign body
3. Cervix – cervicitis, cervical malignancy, cervical erosion, decubitus ulcer
4. Uterus – senile endometritis, tubercular endometritis, endometrial carcinoma
5. Fallopian tube – malignancy
6. Ovary – benign and malignant ovarian tumor
7. Urinary tract – urethral caruncle, papilloma and carcinoma of the bladder
8. Bowel – bleeding from hemorrhoids, anal fissures, rectal cancer are misleading
9. DUB, metropathia hemorrhagica
10. Hypertension, blood dyscrasia
11. Indiscriminate or prolonged use of estrogen unopposed by progestogens
12. HRT when applied cyclically.

➢ **Clinical Features**

1. History
   a. Age of menarche and menopause
   b. History of taking estrogen
   c. History of prolapse
   d. Abdominal pain and foul-smelling discharge are noticed in malignant tumors
   e. Bladder and bowel symptoms.

2. Examination
   a. Blood pressure
   b. General examination
   c. Abdominal palpation
   d. Speculum and bimanual examination.

➢ **Investigations –** Excluding malignancy is the main aim.

1. Blood count and smear – to reveal blood dyscrasia
2. Blood sugar levels
3. Cervical cytology – for cervical lesion
4. Endometrial study – endometrial sample can be obtained by:
   a. D&C
   b. Uterine cavity aspiration
   c. Vibra aspirator
   d. Isaac's aspirator
   e. Pipelle aspirator
   f. Gravlee's jet washer
5. Hysteroscopic inspection and selective biopsy - gold standard in diagnosis of endometrial lesion
6. Ultrasound, CT, MRI – endometrial thickness and irregularity and pelvic tumor. These are useful pre-operative investigations and can detect the extent of spread of the tumour to the myometrium and lymph nodes
7. Diagnostic laparoscopy – to study the nature of the tumour and its spread
8. Cystoscopy and proctoscopy, if necessary.

➢ **Management**

1. Treat the cause
2. When no cause is found:
   a. If there has been only one bout of bleeding, the patient should be kept under observation. About 80% of these cases do not bleed again.
   b. If the woman continues to bleed, or bleeding recurs, it is advisable to perform a laparotomy. An undiagnosed small tumor may be discovered and dealt with appropriately. Otherwise, abdominal hysterectomy with bilateral salpingo-oophorectomy (BSO) should be performed. The specimen should be sent for histopathological study.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. Define menopause. Discuss the management of menopause. 2. Define and describe the causes, investigations and management of postmenopausal bleeding. 3. What are the causes of postmenopausal bleeding? Discuss the methods used to rule out carcinoma cervix. 4. What are the causes of postmenopausal bleeding? How will you diagnose and treat a case of stage I and II endometrial cancer. 5. Discuss causes of postmenopausal vaginal bleeding. Describe staging of endometrial cancer.
Short Essays	1. Artificial menopause. 2. Climacteric symptoms. 3. Menopausal symptoms. 4. Problems (complications) of menopause. 5. Advantages of hormone replacement therapy in menopause. 6. HRT for menopausal symptoms. 7. Contraindications for hormone replacement therapy. 8. Complications of menopause. 9. Postmenopausal bleeding. 10. Etiology of postmenopausal bleeding. 11. Investigations in a case of post- menopausal bleeding. 12. Management of post menopausal bleeding.
Short Answers	1. Advantages of HRT in menopause. 2. Enumerate causes of postmenopausal bleeding. 3. Treatment of menopausal hot flushes. 4. Complications of menopause. 5. Osteoporosis. 6. Define menopause and climacteric symptoms. 7. Symptoms and signs of menopause. 8. Hot flushes in women. 9. Risk factors for postmenopausal osteoporosis. 10. Hormone replacement therapy 11. Non-hormonal treatment of menopause. 12. Differential diagnosis/causes of post menopausal bleeding. 13. How would you counsel a patient for HRT?

Gynecological Diagnosis

CHAPTER 6

DIFFERENTIAL DIAGNOSIS OF PELVIC MASS

- **Uterus, Asymmetrical**
  - Leiomyomata
  - Leiomyosarcoma.
- **Uterus, Symmetrical**
  - Pregnancy
  - Adenomyosis
  - Endometrial cancer
  - Imperforate hymen
  - Hematometra, pyometra.
- **Adnexal, Ovarian**
  - Corpus luteal cyst
  - Follicular cyst
  - Theca lutein cyst
  - Endometrioma
  - Inflammatory cyst (tubo-ovarian abscess)
  - Luteoma of pregnancy
  - Polycystic ovary
  - Benign neoplasms
    - dermoid cyst (most common)
  - Malignant neoplasms
    - granulosa cell tumor (most common)
    - metastatic lesions (e.g., Krukenberg's tumor from stomach).

- **Adnexal, Non-ovarian**
  - Gynecological
    - ectopic pregnancy
    - paratubal cysts
    - pyosalpinx / hydrosalpinx
    - leiomyomata or fibroids
    - primary fallopian tube neoplasms
  - Gastrointestinal
    - appendiceal abscess
    - diverticular abscess
    - diverticulosis, diverticulitis
    - carcinoma of rectum/colon
  - Genitourinary
    - pelvic kidney
    - carcinoma of the bladder.

PELVIC EXAMINATION

- The patient should be asked to lie supine on the examining table and place her feet in stirrups.
- ***Inspection*** of the perineum involves assessment of the hair pattern, skin, presence of lesions (vesicles, warts), evidence of trauma, hemorrhoids, and abnormalities of the perineal body. Genital prolapse can be assessed by gently separating the labia and inspecting the vagina while the patient bears down (Valsalva maneuver).
- ***Spec l m e amination*** begins by choosing the appropriate type and size of speculum, inserting the blades through the introitus and guiding the tip in a downward motion toward the rectum. The blades are opened to reveal the cervix. The vaginal canal should be examined for erythema, lesions, or discharge. The cervix should be pink, shiny, and clear.
- ***iman al e amination*** Two fingers of the gloved hand are introduced into the vagina keeping the thumb off-centre to avoid the clitoris, which is tender to palpation. With the fingers in the posterior fornix, the other hand is placed on the lower abdomen and is the working hand during the pelvic examination. Downward pressure on the abdominal hand should trap the uterus between the hands allowing a determination of its size, regularity, whether it is ante- or retroverted, presence of tenderness and the mobility of the uterus. Palpation for the adnexa commences at the anterior superior iliac spine on each side, using downward pressure to trap the adnexa between the examining hands.
- ***Recto aginal e amination*** is especially valuable when pelvic organs are positioned in the posterior cul-de-sac, in preoperative planning, and in assessment of gynecologic cancers.
- ***Rectal e amination*** performed separately and circumferentially with the examining finger can rule out distally located colorectal cancers. It can also be useful to differentiate between an enterocele and a rectocele.

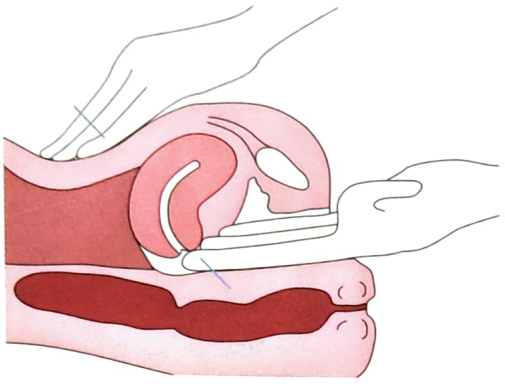


Fig. 6.1: Bimanual examination of the uterus

Fig. 6.2 : Bimanual examination of the adnexa

- Index + middle fingers of the dominant hand are placed in the posterior fornix
- The uterus is elevated by pressing up on the cervix and delivering to the abdominal hand
- The position, size, shape, consistency and mobility of the uterus is noted

- The two fingers of the vaginal hand are moved into the deep right vaginal fornix
- The abdominal hand is placed just medial to the anterior superior iliac spine
- The two hands are brought as close together as possible and the adnexa is palpated with a sliding motion

PAPANICOLAOU TEST (PAP TEST)

- Surface biopsy for detection of cervical cancer. It is a screening test only.
- All women over the age of 35 should undergo an annual check-up with Pap test.
- Can diagnose pre malignant and malignant changes.
- Can also diagnose local conditions. Viral infections like condyloma acuminata and herpes can be detected.
- Positive test requires further investigations like colposcopy, cervical biopsy, and fractional curettage.
- Pap test can detect 98% of cervical cancers and 70% of endometrial cancers
- Single test yields 10-15% false negative reading, which is reduced to only 1% with repeated tests.
- False positive finding is reported in presence of infection.
- Malignant cells are hyperchromatic with a great increase in chromatin content. The nuclei vary in size and there is usually only a small amount of cytoplasm in the undifferentiated malignant cell. The nucleus: cytoplasm ratio is increased in malignant cells.
- **Classifications**

Grade	Papanicolaou	WHO	Bethesda
1	Normal cells	Normal	Normal
2	Slightly abnormal, suggestive of inflammatory change	Inflammatory	Inflammatory (HPV & ASCUS)
3	Abnormal, usually requires biopsy	CIN I	Low SIL

4	Distinctly abnormal, definitely requires biopsy	CIN II & CIN III & Carcinoma-in-situ	High SIL
5	Malignant cells	Squamous cell carcinoma	Squamous cell carcinoma

- **Procedure**

1. Patient is placed in dorsal position and labia is separated.(Pap test to be done prior to vaginal examination)
2. Insert the speculum into the vagina (without gel), letting the speculum follow the path of least resistance. Keep the speculum blades closed until the speculum is completely inserted.
3. Open the speculum and usually the cervix is immediately visible. If not, the cervix is usually just below the lower blade or just above the upper blade.
4. Lock the blades in the open position, wide enough apart to allow complete visualization of the cervix but not too far open as to be uncomfortable for the patient.
5. The Ayer's spatula is specially designed for obtaining Pap smears. The concave end (curving inward) fits against the cervix, while the convex end (curving outward) is used for scraping vaginal lesions or sampling the "vaginal pool," the collection of vaginal secretions just below the cervix.
6. The concave end of the spatula is placed against the cervix and rotated in circular fashion so that the entire area around the cervical opening (os) is sampled.
7. Spread the sample taken from the cervix on a glass slide. Try to make the smear as thin as possible since this makes it easier for the pathologist to read.
8. Immediately spray the glass slide with cytological fixative or dip the slide in a jar with equal parts of 95% ethylate and ether.
9. Next, use a "cytobrush" to sample the endocervical canal, the inside of the opening leading into the uterine cavity.
10. Push the cytobrush into the canal, no deeper than the length of the brush (1.5 cm - 2.0 cm). Rotate the brush 180 degrees (half a circle) and pull the cytobrush straight out.
11. Smear the sample on another slide, spreading the material evenly over the slide. Spray with fixative immediately and examine it under a microscope.

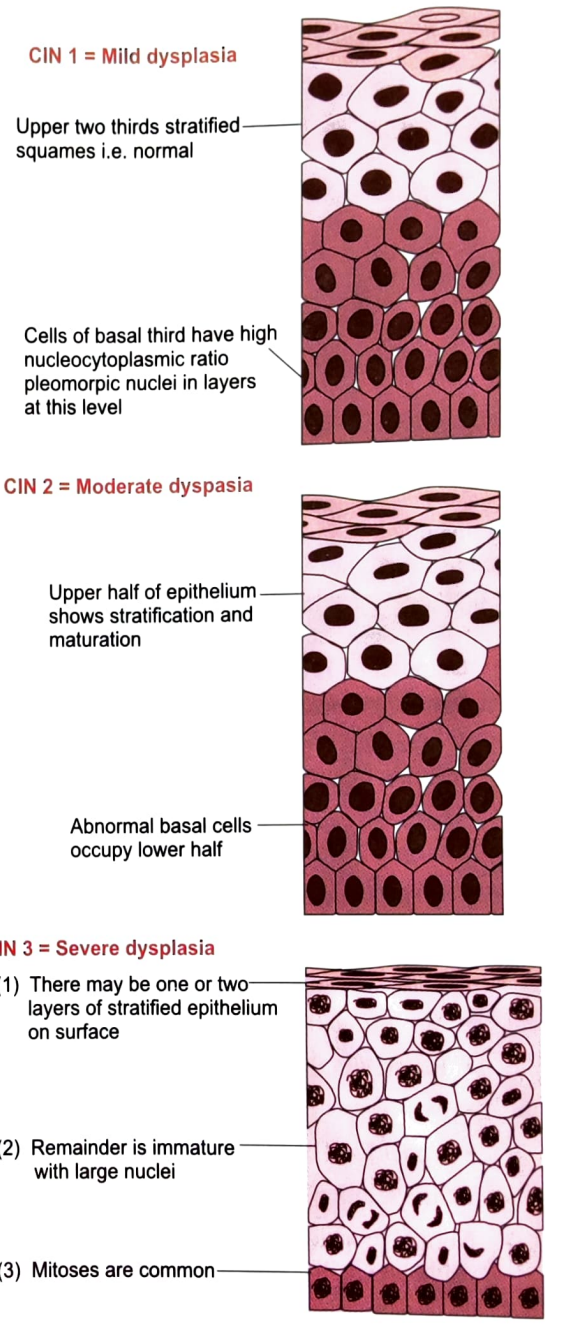

CIN 1 = Mild dysplasia
Upper two thirds stratified squames i.e. normal
Cells of basal third have high nucleocytoplasmic ratio pleomorpic nuclei in layers at this level
CIN 2 = Moderate dyspasia
Upper half of epithelium shows stratification and maturation
Abnormal basal cells occupy lower half
CIN 3 = Severe dysplasia
(1) There may be one or two layers of stratified epithelium on surface
(2) Remainder is immature with large nuclei
(3) Mitoses are common

Fig. 6.3: Various degrees of dysplasia in CIN

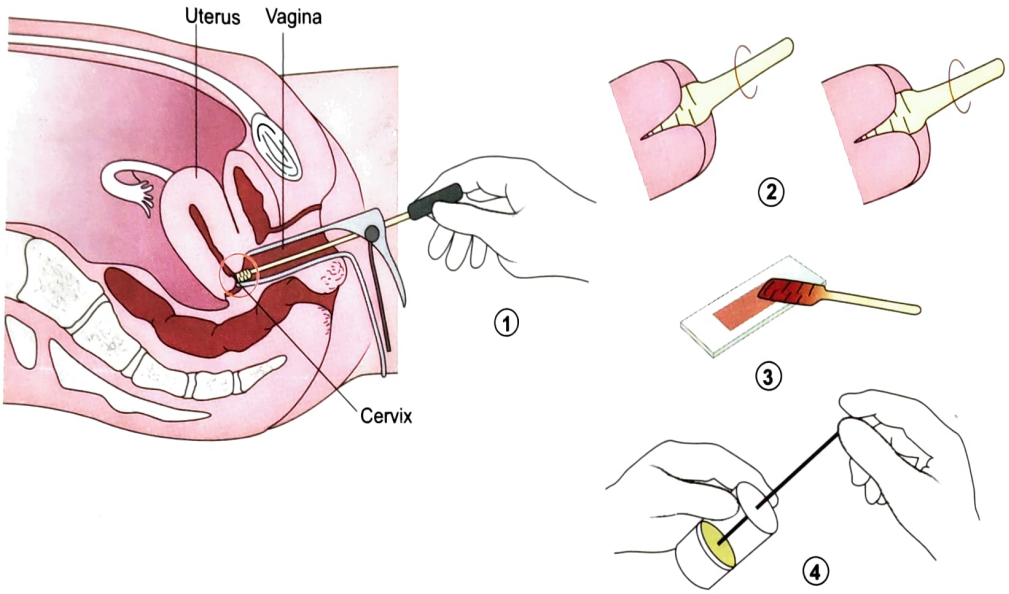


Fig. 6.4: Procedure of Pap test

COLPOSCOPY

- Colposcopy is an outpatient office procedure. It uses a binocular, short focal-length instrument with a built-in light source to look at the cervix through a speculum.
- A speculum is passed and the cervix visualized as for a smear. 4% Acetic acid is painted onto the surface of the cervix with a cotton wool swab.
- Abnormal cells at the squamo-columnar junction will stain white with this liquid because of their increased glycogen content (Acetowhite).
- The speed and the density of the white color are proportional to the degree of abnormality.
- The abnormal areas are noted and drawn in diagramatic form in the notes.
- Additional features that can be looked for that have been associated with microinvasive disease are:
  - Mosaicism (tile like formation of the cells);
  - Punctation (small dots on the surface of the cervix);
  - New vessel formation (using a green light filter);
  - Upper limit of the abnormality inside the cervical canal must be seen.
- Iodine solution is then painted onto the cervix.
- Normal squamous epithelium will stain dark brown with this solution while abnormal cells and normal columnar epithelium will remain unstained.
- This delineates the area more accurately but does not give an idea of how severe the lesion may be so acetic acid should always be used first.
- Once the lesion has been defined a biopsy is taken.

➢ **Uses of Colposcopy:**

Diagnostic use

- Provides a magnified view of the surface structures of the vulva, vagina and cervix
- Special green filters allow better visualization of vessels
- Application of 1% acetic acid wash dehydrates cells and reveals white areas of increased nuclear density (abnormal) or areas with epithelial changes
- Biopsy of visible lesions or those revealed with the acetic acid wash allows early identification of dysplasia and neoplasia.

Therapeutic use

- Cryotherapy
- Tissue destruction by freezing
- For dysplastic changes, genital warts
- Laser
- Cervical conization
- Removes the cervical transformation zone and areas within the endocervical canal
- Methods include cold knife, laser excision, or electrocautery.

➢ **Cold knife cone biopsy** is a minor outpatient surgical procedure performed in the operating room under either local or general anesthesia. It is a diagnostic test that examines the histology of cervical lesions. A cone-shaped tissue specimen is obtained with a scalpel by performing a circumferential incision of the cervix with a diameter that is wider at the cervical os and narrower toward the endocervical canal. This tissue is sent to pathology for histologic diagnosis.

➢ **LEEP** (Loop Electrosurgical Excision Procedure) is a minor outpatient surgical procedure performed under local anesthesia. This technique is used for diagnosing and treating cervical dysplasia. An electric current is passed through a thin wire loop to remove abnormal cervical tissues.The heated loop seals off blood vessels as it cuts. Advantages are low cost, high success rate, and ease of use.

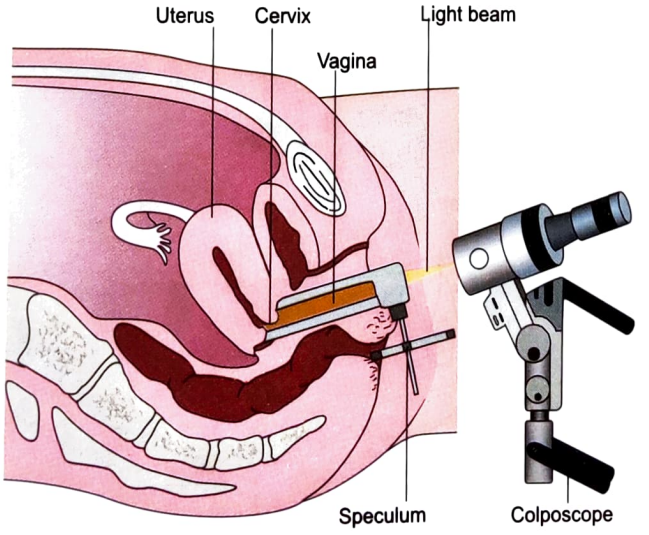


Fig. 6.5: Colposcopy

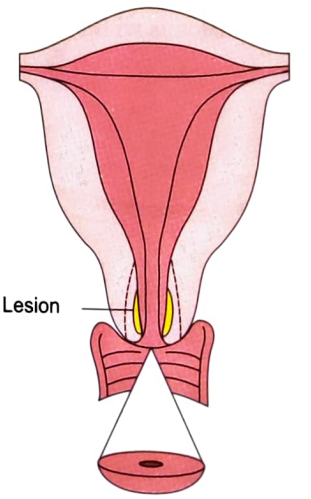


Fig. 6.6: Cold knife cone biopsy

ENDOMETRIAL BIOPSY

- It is a method of sampling the endometrium. The sample obtained is smaller than D&C.
- **Indications**

1) Patients with infertility to check if ovulation is occurring (secretory endometrium indicates ovulation).
2) Patients with abnormal vaginal bleeding to rule out endometrial hyperplasia or cancer.
3) Patients with menorrhagia who are >40 yrs of age to exclude endometrial cancer, a small sample is taken for rapid diagnosis prior to a formal D&C.
4) Patients with post-menopausal bleeding.

- **Procedure**

The patient is placed in lithotomy position, perineum is cleaned and draped.

A Sim's speculum is introduced into the vagina and the posterior vaginal wall retracted.

The anterior cervical lip is held with a vulsellum.

Endometrial biopsy curette is introduced into the cervix.

A 20 cc syringe is used to create suction and aspirate the curettings which are usually inspected and sent for HPE and AFB culture.

- **Advantages**

Does not require dilatation of cervix and hence avoids associated complications.

Curettage with plastic cannula decreases risk of perforation.

- **Instruments**

The Novak curette is a thin metallic tube with a side opening at the tip; suction with attached syringe can be applied to help to remove tissue.

The Pipelle is a more flexible plastic tube with a side opening at the tip. A smaller tube (internal piston) inside the Pipelle is withdrawn to create suction. Pipelle is inserted through the cervix to the full depth of the endometrial cavity. Piston is withdrawn and the sheath is rotated to sample the endometrium.

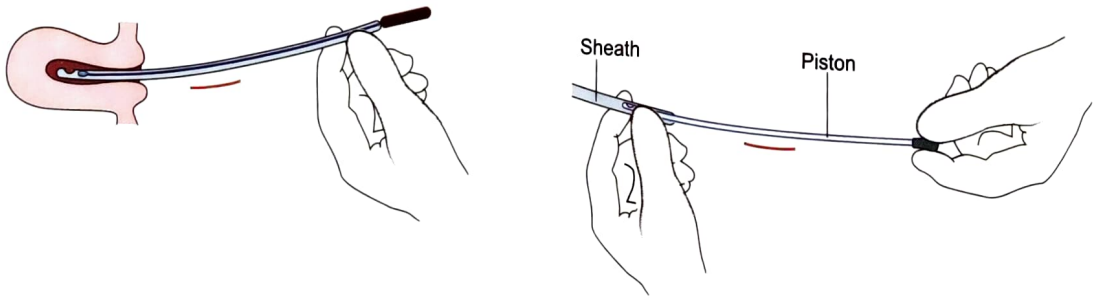


Fig. 6.7: Endometrial biopsy

PREVIOUS EXAMINATION QUESTIONS

Short Essays	1. Differential diagnosis of pelvic mass. 2. Differential diagnosis of mass felt through right fornix. 3. Bimanual pelvic examination. 4. Indications for rectal examination in gynecology. 5. Colposcope – principles and indications 6. Papanicolaou's smear/Pap smear. 7. Method of taking a Pap smear and sending to cytology laboratory.
Short Answers	1. Cervical biopsy. 2. Indications for endometrial biopsy. 3. Causes of fullness in pouch of Douglas. 4. Causes of symmetrical enlargement of uterus. 5. Rectal examination in gynecology (indications). 6. Pap smear. 7. Endometrial biopsy.

Malformations of the Female Generative Organs

CHAPTER 7

HEMATOCOLPOS

- **Definition** – Collection of blood in the vagina due to atresia of lower vagina (hymen).
- **Etiology**
  1. **Congenital** – Imperforate hymen
     - It is mostly unnoticed until the girl attains the age of 14-16 years.
     - As the uterus is functioning normally, the menstrual blood is pent up inside the vagina behind the hymen (cryptomenorrhea).
     - Depending upon the amount of blood accumulated, it first distends the vagina (hematocolpos).
     - The uterus is next involved and the cavity dilated (hematometra).
     - In the late and neglected stages, the tubes may also be distended after the fimbrial ends become closed by adhesions (hematosalpinx).
  2. **Acquired –** Due to adhesions following infection.
- **Clinical Features**
  1. Colicky abdominal pain which is often cyclic
  2. Menarche has not yet set in
  3. Secondary sexual characteristics are well-developed
  4. Pressure symptoms
     a. Difficulty during micturition
     b. Urinary retention
  5. Hypogastric or suprapubic bulge which may cause discomfort on palpation
  6. On separation of the labia, a bluish bulging membrane is seen
  7. Rectal examination reveals bulge of the vagina anteriorly.
- **Complications**
  1. Hematometra
  2. Hematosalpinx
  3. Pelvic endometriosis.

- **Investigations** – Pelvic ultrasound
- **Treatment**
  1. Cruciate incision is made on the hymen
  2. Surgical excision of the hymen
  3. Drainage of the hematocolpos.

MULLERIAN DUCT ANOMALIES

- Mullerian duct anomalies result from defective fusion, canalization or absorption of the median septum of the female reproductive system during embryonic development.
- **Classification**
  1. Class I – Segmental Mullerian agenesis or hypoplasia
     a. Vaginal
     b. Cervical
     c. Fundal
     d. Tubal
     e. Combined.
  2. Class II – Unicornuate uterus
     a. With a rudimentary horn
        1) With a communicating endometrial cavity

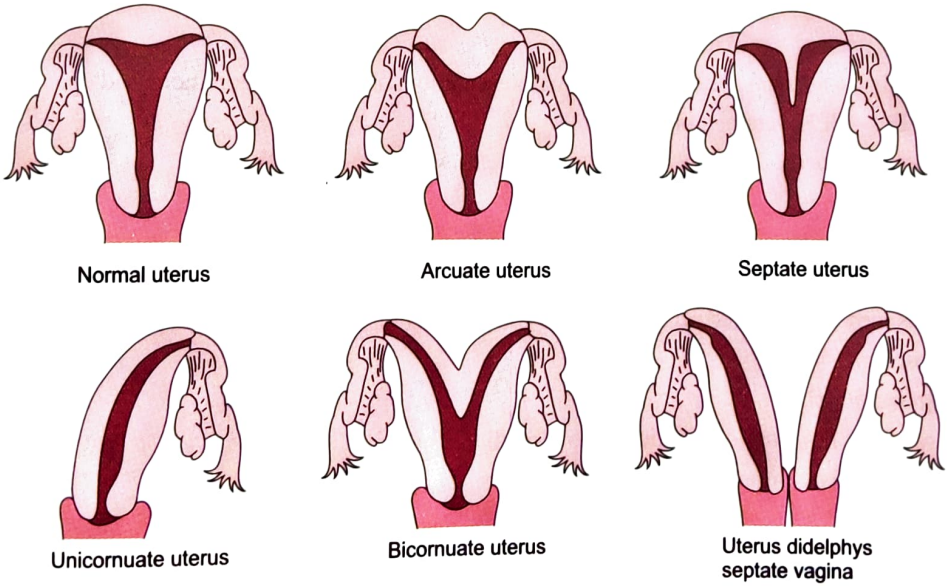


Fig. 7.1: Mullerian duct anomalies

2) With a non-communicating cavity
3) With no cavity

b. Without any rudimentary horn.

3. Class III – Uterus didelphys.
4. Class IV – Bicornuate uterus
   a. Complete upto the internal os
   b. Partial
   c. Arcuate.
5. Class V – Septate uterus
   a. With a complete septum
   b. With an incomplete septum or uterus subseptus.
6. Class VI – Uterus with internal luminal changes.

➢ **Clinical Features**

1. Many cases are asymptomatic
2. Symptomatic patients may present with:
   a. Abortions
   b. Preterm births
   c. LBW babies
   d. Malpresentations
   e. Retained adherent placenta
   f. PPH.

➢ **Adverse effect on pregnancy**

1. During pregnancy – mid trimester abortion
2. During labor – abnormal uterine action, malpresentation, obstructed labor, increased operative interference
3. After labor – PPH, retained placenta
4. Fetus – prematurity.

➢ **Diagnostic Methods**

1. Bimanual palpation
2. Hysterosalpingography
3. Hysteroscopy – gold standard in diagnosis and therapeutic management
4. Laparoscopy
5. Ultrasound of pelvis
6. Ultrasound of renal region for kidney abnormality
7. MRI of the pelvic organs.

➢ **Treatment**

1. Strassman utriculoplasty operation – Transverse fundal incision is made for reunification of the uterine cavity. Done in cases of bicornuate uterus.
2. Jones' operation – Wedge resection of a part of the fundus of the uterus along with the septum followed by uniting the 2 horns vertically. Done in cases of septate uterus.
3. Tomkins operation – It consists of incising the fundus in the midline, exposing the septum and excising it.

followed by reuniting the 2 parts in the midline. Done in cases of septate uterus.

4. Hysteroscopic resection of the septum - Done with a resectoscope or CO_2 laser. Asherman syndrome and adherent placenta are the likely complications.
5. A rudimentary horn needs excision of the horn to avoid hematometra, ectopic pregnancy and endometriosis.

PREVIOUS EXAMINATION QUESTIONS	
Short Essays	1. Hematocolpos clinical features and treatment. 2. Write in brief about congenital abnormalities of uterus and their significance. 3. Uterus didelphys. 4. Classify Mullerian duct anomalies. 5. Hematometra. 6. Imperforate hymen.
Short Answers	1. Diagnosis and treatment of urogenital atrophy. 2. Septate uterus. 3. Uterine anomalies classify. 4. Causes of pelvic hematocele. 5. Imperforate hymen. 6. Bicornuate uterus. 7. Problems of bicornuate uterus.

Sex and Intersexuality

CHAPTER 8

TURNER'S SYNDROME

- Turner's syndrome is characterized by hypogonadism in phenotypic females.
- It results from complete or partial monosomy of X-chromosome.
- Karyotype – 45 XO.
- Turner's syndrome is the single most important cause of primary amenorrhea.
- **Clinical features**

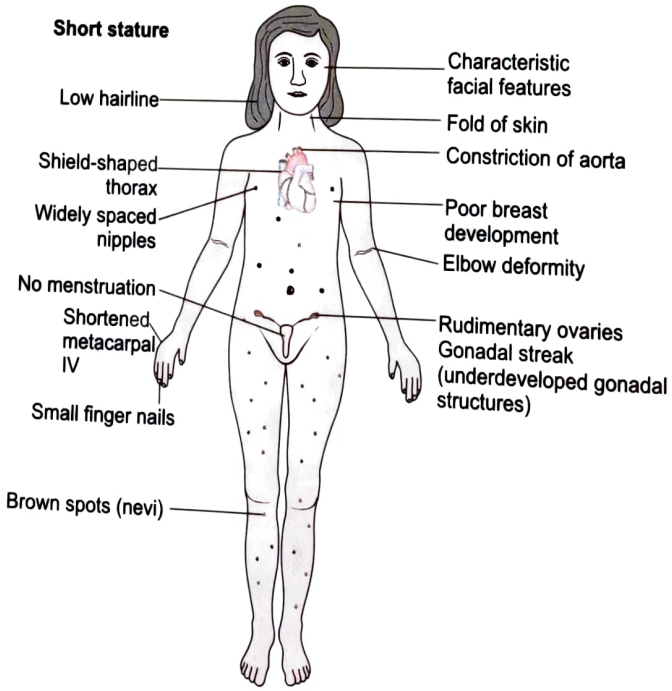


Fig. 8.1: Turner's syndrome

➢ **Treatment**

There are two main medications given to girls with Turner syndrome.

1. Human growth hormone, used to increase the girl's growth rate and help her become taller.
2. Estrogen, to replace the estrogen which would normally have been produced by the ovaries. Progesterone is also given when the girl grows older, to help her have a normal monthly menstrual cycle.

KLINEFELTER'S SYNDROME

- ➢ Klinefelter's syndrome is characterized by hypogonadism in phenotypic males.
- ➢ It results from an extra X-chromosome.
- ➢ Karyotype – 47 XXY.
- ➢ **Clinical features**

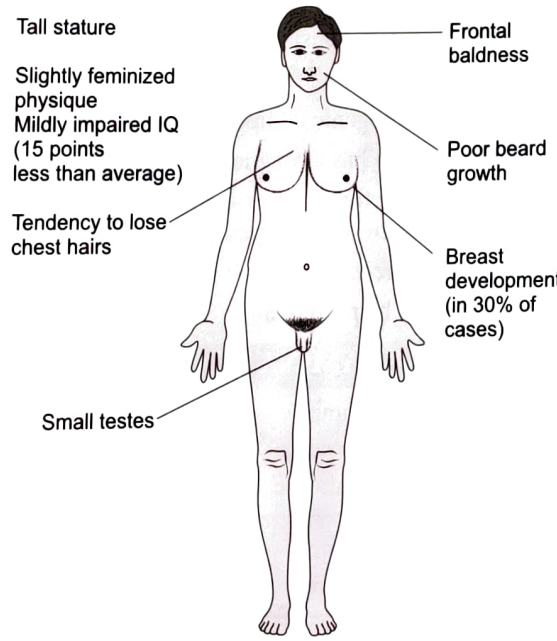


Fig. 46.2: Klinefelter's syndrome

➢ **Treatment**

- Testosterone replacement therapy – Testosterone may be given as an injection or with a gel or patch on the skin.
- Breast tissue removal by surgical methods.
- Speech and physical therapy to overcome problems with speech, language and muscle weakness.
- Psychological counselling.

CAUSES OF HIRSUTISM

➢ **Definition** – Hirsutism is excessive male-pattern hair growth in a woman on the upper lip, chin, chest, abdomen, back, and proximal extremities.

➢ **Pathophysiology**

- Hirsutism involves the conversion of vellus hair (fine, nonpigmented hair) to terminal hair (coarse, dark hair) within the hair follicle. This conversion is under the influence of androgens.
- It can be caused by either an increased level of androgens or an oversensitivity of hair follicles to androgens.
- These conditions include polycystic ovary syndrome (PCOS) (the most common cause), congenital adrenal hyperplasia, Cushing's disease, acromegaly, tumors in the ovaries, insulin resistance and obesity (as there is peripheral conversion of androgens to estrogen in these patients).

➢ **Laboratory Tests**

The primary purpose of these tests is to identify elevated free androgens.

- Dehydroepiandrosterone sulfate (DHEAS)- is produced only in the adrenal glands. A markedly elevated DHEAS is consistent with an adrenal tumor.
- 17-OH progesterone – is elevated in late onset congenital adrenal hyperplasia (CAH).
- Testosterone – is produced by both the ovary and the adrenal glands. A mildly elevated level is suggestive of PCO syndrome. A markedly elevated level is consistent with an ovarian tumor.

➢ **Management**

- Spironolactone – antialdosterone, antiandrogenic compound.
- Cyproterone acetate – a progestin that also has strong antiandrogenic action.
- Metformin – antihyperglycemic drug used for diabetes mellitus. However, it is also effective in treatment of hirsutism associated with insulin resistance (e.g., polycystic ovary syndrome).
- Eflornithine – blocks putrescine that is necessary for the growth of hair follicles.
- Other methods – epilation, waxing, laser hair removal.

PREVIOUS EXAMINATION QUESTIONS

Short Essays	1. Turner's syndrome. 2. Diagnosis of Turner's syndrome. 3. Klinefelter's syndrome.
Short Answers	1. Turner's syndrome. 2. Causes of hirsutism.

Diseases of the Vulva

CHAPTER 9

PRURITUS VULVA

- It is an itching sensation with a desire to scratch the vulva.
- Vulval irritation is not the same as pruritus, but it is a painful condition associated with burning. Prolonged or severe pruritis can eventually lead to vulval irritation through scratching and abrasions.
- **Etiology**

1. Vulval
   - i. Parasitic:
     - a. Scabies
     - b. Pediculosis
   - ii. Chronic dystrophies:
     - a. Leucoplakia
     - b. Lichen sclerosis
   - iii. Radiation vulvitis
   - iv. Vulval diseases:
     - a. Condyloma acuminata
     - b. Granulomas
     - c. Behcet's syndrome
     - d. Paget's disease.
2. Vaginal discharge due to:
   - a. Trichomonas vaginalis
   - b. Candida albicans.
3. Cervical
   - a. Cervicitis
   - b. Erosion.

4. Anal
   a. Threadworm infestation.
5. Urinary
   a. Bacilluria
   b. Glycosuria
   c. Acidic urine
   d. Incontinence.
6. General diseases
   a. Diabetes
   b. Jaundice
   c. Cirrhosis.
7. Nutritional
   a. Iron deficiency anemia
   b. Vitamin A deficiency
   c. Vitamin B_2 deficiency.
8. Allergy
   a. Drugs
   b. Soap, detergents, antiseptics, phenol, etc.
9. Contact dermatitis.
10. Psychological – psychoneurosis due to stress.

➢ **Treatment** – The cause should be investigated systematically and treated.

1. Antihistamines and sedation – symptomatic treatment
2. Hydrocortisone ointment – local application
3. Estrogen cream – in kraurosis vulva due to menopausal changes
4. Nystatin cream – in fungal infection
5. Imidazole group of antifungal drugs (e.g. miconazole)
6. Metronidazole – given orally; specific for Trichomonas
7. Cream made of zinc oxide and olive oil or cod liver oil – if the skin is hard and tends to crack
8. Injection of absolute alcohol subcutaneous – breaks the scratch habit
9. Human leucocyte interferon ointment – applied 4 times a day for 5 weeks
10. Systemic IM interferon – 2 million units daily for 10 days
11. Ball's operation – now rarely performed; comprises division of cutaneous nerves by circular incision around the vulva; effect lasts for 3-6 months.

LEUKOPLAKIA VULVA

➢ It is a clinical term, used to refer to a diverse group of vulvar skin lesions.

➢ While they may have a variety of aetiologies and histopathological patterns, these lesions present as opaque, white, plaque like mucosal thickenings that are sometimes pruritic and scaly.

➢ They appear white to the naked eye even prior to application of 3% acetic acid used in colposcopy.

➢ Biologically, these lesions range from benign to premalignant to malignant. This group of lesions include:

- Vitiligo;
- Inflammatory dermatosis (e.g., psoriasis, chronic dermatitis);
- Carcinoma in situ, Paget's disease of vulva, invasive carcinoma; and
- A variety of lesions of unknown aetiology which cannot be properly classified.

DIFFERENTIAL DIAGNOSIS OF VULVAL ULCERS

Infectious	Non-infectious
Venereal	Aphthous ulcers
Herpes simplex (HSV)	Behcet's disease
Chancroid	Crohn's disease
Granuloma inguinale	Fissures
Lymphogranuloma venereum	Tumors – BCC
Syphilis	
Human immunodeficiency virus	
Non-Venereal	
EBV	
Mycoplasma pneumoniae	

GENITAL WARTS (Condyloma accuminata)

- They are caused by human papilloma virus.
- HPV subtypes 6 and 11 are more commonly associated with external genital warts whereas HPV types 16, 18, 31, 33, 35 and 39 with genital cancer.
- Clinical features – Asymptomatic verrucous papules on the genitalia.
- Diagnosis – Visualisation of the lesion. If doubtful, a biopsy is done.
- Treatment:
  - Spontaneous resolution seen in 10-20% cases.
  - Associated infections should be eradicated and counselling regarding the recurrence to be done.
  - Application of purified podophyllin resin, imiquimod 5% cream, 10-25% podophyllin solution, trichloro - acetic acid (safe in pregnancy), topical 5-flurouracil.
  - Recurrent or resistant warts can be treated with interferon injection.
  - Cryotherapy is used for multiple cervical lesion
  - Surgical excision for large perianal lesions.

VULVECTOMY

- **Definition** – Vulvectomy refers to a gynecological procedure in which the vulva is partly or completely removed.
- **Types**

  1. Skinning vulvectomy removes the top layer of skin

2. Simple vulvectomy removes multiple layers of skin and tissue
3. Partial vulvectomy removes a part of the vulva, as well as some nearby tissue and lymph nodes
4. Radical vulvectomy removes the entire vulva, including nearby tissue and lymph nodes.

➤ **Indications**

1. Vulvar intraepithelial neoplasia (VIN)
2. Paget disease of the vulva
3. Pigmented lesions and melanomas of limited size
4. Vulvar dermatologic conditions.

A simple vulvectomy is performed for benign or premalignant conditions of the vulva that are extensive or multifocal and are not amenable to removal with local excision alone.

PREVIOUS EXAMINATION QUESTIONS	
Long Essays	
Short Essays	1. Leukoplakia vulva. 2. Pruritis vulvae causes and treatment.
Short Answers	1. Pruritis vulva – gynecological causes. 2. Diagnosis and treatment of leukoplakia vulva. 3. Causes of pruritis vulvae. 4. Genital warts – clinical features and treatment. 5. Leukoplakia vulvae. 6. Indications for simple vulvectomy. 7. Different diagnosis of vulval ulcer.

Diseases of the Vagina

CHAPTER 10

LEUCORRHEA

- **Definition –** Excessive secretion of normal vaginal discharge.
- The term leucorrhea should fulfil the following criteria:
  1. Excess secretion is evident from persistent vulval moistness or staining of the undergarments (brownish yellow on drying) or need to wear a vulval pad
  2. Non-purulent and non-offensive
  3. Non-irritant (never causes pruritus).
- **Causes**
  1. Physiological
     a. At birth – due to effect of placental estrogen on genital organs
     b. At puberty – due to increased vascularity of pelvic organs
     c. During ovulation – due to increased estrogen
     d. Premenstrual – due to pelvic congestion and increased mucous secretion by hypertrophied endometrial glands
     e. During pregnancy – due to increased hormone levels
     f. During sexual excitement – due to increased secretions from the Bartholin's glands.
  2. Pathological
     i. Cervical leucorrhea – non-infective cervical lesion may produce excessive secretion which pours out at the vulva.
        a. Cervical erosion
        b. Chronic cervicitis
        c. Mucus polyp
        d. Ectropion.
     ii. Vaginal leucorrhea – increased vaginal transudation occurs in conditions associated with increased pelvic congestion.
        a. Uterine prolapse

b. Acquired retroverted uterus
c. Chronic pelvic inflammation
d. OCP's
e. Vaginal adenosis.

- **Diagnosis**

1. The excessive discharge has got relevance with the above conditions
2. Discharge is non-offensive and non-irritant
3. General examination may reveal ill health
4. Vulval inspection reveals:
   a. White or creamy discharge
   b. No evidence of pruritus
5. Bimanual and speculum examinations reveal:
   a. A negative pathology
   b. Associated pelvic lesions causing cervical or vaginal leucorrhea
6. Microscopic examination of the discharge – pus cells are indicative of infection
7. If pus cells are detected, further investigations are carried out to identify the organism from the discharge. Neoplasm and foreign body are excluded.
   a. Hanging drop preparation
   b. Gram stain
   c. Culture.

- **Treatment**

1. Improvement of general health
2. Sympathetic attitude towards the ailments. Anxiety should be removed.
3. Cervical lesions – surgical treatment such as electrocautery, cryosurgery, trachelorrhaphy, etc.
4. Pelvic lesions – appropriate therapy specific for the pathology.
5. OCP users may have to temporarily discontinue using it.
6. Local hygiene has to be maintained meticulously.

CANDIDIASIS (MONILIASIS)

- **Causative Organism –** Candida albicans.
- **Predisposing Factors**

1. It flourishes in an acidic medium with abundant supply of carbohydrate. Therefore, it is common in pregnancy and diabetes.
2. Hormonal contraceptive pills, vaginal douching.
3. Immunosuppression – HIV or immunosuppressive therapy.
4. Prolonged use of antibiotics.

- **Symptoms**

1. Profuse, curdy discharge (odorless)
2. Intense pruritus

3. Vulval irritation
4. Dysuria
5. Burning sensation.

- **Signs**
  1. Inflammation of the vulva red and edematous
  2. White patches of cheesy curdy material adherent to the vagina, which when removed leave multiple petechiae-like hemorrhagic areas
  3. Discharge is "cottage cheese like".
- **Investigations**
  1. Examination of KOH mount – candidal hyphae and buds stand out in prominence along with spores.
  2. Discharge culture on Sabouraud's medium – rounded colonies, 1-2 mm in diameter, within 48-72 hours, with yeast-like odor.
  3. Discharge culture on Nickerson's medium – brown-black colonies in 48-72 hours
- **Treatment**
  1. Advice regarding personal hygiene
  2. Avoidance of synthetic undergarments
  3. Finger nails should be clipped short
  4. Oral drugs
     i. Fluconazole 150 mg single dose
  5. Vaginal
     i. Nystatin – 1-2 vaginal pessaries for 10-14 days
     ii. Imidazole derivatives (miconazole, clotrimazole, terconazole) – vaginal pessaries or creams
  6. Treatment of the cause – control of diabetes, stoppage of OCP, etc.
- **Recurrent vulvovaginal candidiasis:** 4 or more episodes per year.

NON-SPECIFIC VAGINITIS / BACTERIAL VAGINOSIS

- In this group of disorders, a variety of mixed pathogens are recoverable on smear and culture, e.g. Staphylococcus, Streptococcus, E. coli, etc.
- **Etiology** (Alteration in normal vaginal flora – decreased lactobacilli and increase in other bacteria)
  1. Chemicals, drugs, contraceptives
  2. Douches, pessaries, tampons, foreign bodies
  3. Trauma
  4. Operations – vaginal and cervical.
- **Diagnosis** – based on the presence of 3 of the 4 signs
  1. White milky adherent discharge
  2. pH > 4.5
  3. Whiff (sniff) test positive (fishy odor on adding 10% KOH)
  4. Clue cells on microscopy, these are vaginal epithelial cells whose surface is stippled with coccobacillus.
- **Complications:** Group B streptococcal infection and bacterial vaginosis have been implicated in late 2nd trimester abortion and preterm labor. Infection with Staphylococcus aureus may result in toxic shock syndrome,

more so associated with the use of tampons.

- **Treatment**
  1. General – improvement of general health
  2. Local
     a. Water dispersible buffered vaginal jelly – to correct the vaginal pH to 4.5
     b. Bactericidal cream such as triple sulpha
     c. Antibiotic pessaries, if organism and sensitivity are known
     d. Diathermy cauterization and conization – to eliminate infection in the genital tract such as chronic endocervicitis
     e. FAS-3 kit – contains fluconazole, azithromycin, and secnidazole.

SENILE VAGINITIS (ATROPHIC VAGINITIS)

- **Definition –** Vaginitis in postmenopausal women.
- **Etiology –** The basic underlying pathology is estrogen deficiency.
  1. Menopause
  2. Premature menopause
  3. Prolonged lactation
  4. Oophorectomy operation.
- **Clinical Features**
  1. Dry vagina
  2. Dyspareunia
  3. Discharge – purulent, slightly blood-tinged
  4. Urinary symptoms – dysuria, frequency
  5. Vagina – inflamed and tender with excoriation of the mucosa
  6. Low grade chronic urethritis
  7. Patchy granular vaginitis, the spots of which are red and bleed easily when swabbed
  8. Infection may spread upwards to cause senile endometritis and pyometra.
- **Diagnosis –** Senile endometritis may co-exist and carcinoma of the endometrium or cervix should be excluded prior to therapy.
  1. Examination under anesthesia
  2. Diagnostic curettage
  3. Cervical cytology or biopsy.
- **Treatment**
  1. Estrogen – to improve the resistance of the vaginal epithelium, raise the glycogen content and lower the vaginal pH
  2. Ethinyl estradiol – 0.01 mg daily for 3 weeks
  3. Pessary containing estrogen
  4. Vaginal cream containing estrogen.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. Define leucorrhea. Discuss the causes and management of white discharge per vagina. 2. How will you investigate and treat a case of 30 year old patient presenting with white discharge per vagina? 3. Define leucorrhea. Mention its causes and describe its management. 4. What are the causes of white discharge in a 30 year old woman? Describe the relevant investigations and treatment.
Short Essays	1. Differential diagnosis of discharge per vagina and their management. 2. Evaluation of leucorrhea. 3. Diagnosis and treatment of Trichomonas vaginalis infection. (Trichomonas theory in next chapter) 4. Etiology, diagnosis and treatment of monilial vaginal infection. 5. Monilial vaginitis. 6. Senile vaginitis diagnosis and management. 7. Vaginal trichomoniasis. 8. Diagnosis and treatment of vaginal candidiasis. 9. Differential diagnosis of discharge per vagina and their management. 10. Causes of leucorrhea in reproductive age group. 11. Give the causes of vaginal discharge. How will you diagnose and manage? 12. Bacterial vaginosis – diagnosis and treatment. 13. Vaginal discharge. 14. Group B streptococcal infection. 15. Trichomonas vaginalis. 16. Metronidazole. 17. Predisposing factors and diagnosis of monilial vaginitis. 18. Management of candidiasis (moniliasis) in gynecology. 19. Treatment of recurrent moniliasis. 20. Treatment of vulvovaginal moniliasis.
Short Answers	1. Bacterial vaginosis – diagnosis and treatment. 2. Sniff test. 3. Treatment of vulvovaginal moniliasis. 4. Management of candidiasis in gynecology. 5. Causes and management of vaginitis. 6. Treatment of vaginal trichomoniasis. 7. Toxic shock syndrome.

Sexually Transmitted Diseases

CHAPTER 11

Spectrum of organisms

- **Bacterial** – These include chancroid, lymphogranuloma venereum, granuloma inguinale, chlamydia, gonorrhea, syphilis.
- **Viral** – These include condyloma acuminatum, herpes simplex, hepatitis B virus, and human immunodeficiency virus.
- **Protozoan** – This includes trichomoniasis.

GONORRHEA

- **Age & Incidence** – In the females, gonococcal infection can occur at any age, but is most commonly seen between menarche and menopause.
- **Causative Organism** – Neisseria gonorrheae.
- **Mode of Transmission**
  1. Sexual contact
  2. May be transmitted accidentally in children.
- **Pathology**
  1. Vaginal squamous epithelium is resistant to gonorrhea
  2. It penetrates the columnar epithelium of Skene and Bartholin's glands, urethra and urethral glands
  3. Spreads along mucous membrane to affect cervix, endometrium and fallopian tube
  4. It may also ascend attached to a sperm
  5. It may spread by bloodstream and form abscesses.
- **Clinical Features** – Local, pelvic, and metastatic
  1. Local
     a. Urethritis – dysuria, purulent discharge, pan-urethral abscess.
     b. Skenitis – pus may be expressed from gland ducts.
     c. Bartholinitis – Bartholin's glands are inflamed and patient complains of marked tenderness and swelling. Pressure on the gland results in the expression of a bead of pus from the duct openings on the inner surface of the labia minora. Bartholin's abscess may rupture through the vaginal epithelium or the skin of the perineum.
     d. Vulvitis – discomfort and swelling of the labia.
     e. Vaginitis – trichomoniasis may co-exist with gonorrhea.

f. Cervicitis – It may be asymptomatic or may cause backache or lower abdominal discomfort. Increased mucopurulent vaginal discharge often seen as coming from the cervical os. The os may be hyperemic and an erosion may be present.

g. Cystitis – mild trigonitis is not uncommon, but renal infection is rare.

h. Proctitis – rectal discharge with discomfort on defecation or rectal bleeding.

i. Pharyngitis – may occur following oral sex.

2. Pelvic

a. In chronic cases, women complain of backache, dull lower abdominal pain, menorrhagia, polymenorrhea, dyspareunia and infertility.

b. Salpingitis with resultant tubo-ovarian abscess, pyosalpinx, hydrosalpinx and possibly pelvic abscess are the known sequelae.

3. Metastatic

a. Gonococcal arthritis

b. Gonococcal septicemia with acute bacterial endocarditis

c. Pericarditis

d. Perihepatitis (Fitz-Hugh-Curtis syndrome)

e. Ophthalmitis

f. Meningitis.

➢ **Investigations**

1. Smears of the urethral and cervical discharge
2. Culture of the discharge on Thayer-Martin medium or McLeod's chocolate agar with 5% CO_2
3. Gonococcal complement fixation test
4. Fluorescent tests.

➢ **Treatment**

1. Penicillin – drug of choice; Procaine penicillin – 4.8 mega units.
2. Probenecid or amoxicillin or ampicillin also effective.
3. Abscesses – drainage of pus before administering antibiotics.
4. Pelvic infection – benzyl penicillin IM 6 hourly for 3 days, which is followed by procaine penicillin daily for 7 days.
5. For PPNG, single IM Inj. 2 g spectinomycin or 1 g cefotaxime.
6. Contact tracing and sexual partner notification.

➢ **Complications**

1. Acute salphingitis/PID – is the most common complication in women. Chronic infection leads to hydro/pyosalphinx
2. Bartholin's gland abscess – commonly unilateral
3. Disseminated infection – characterised by acute arthritis and dermatitis.

SYPHILIS

- **Causative Organism -** Treponema pallidum.
- **Mode of Transmission -** Direct contact with an infectious lesion.
- **Primary syphilis**
  - Painless chancre on vulva, vagina or cervix
  - Painless inguinal lymphadenopathy
  - 3-4 weeks after exposure
  - Serological tests usually negative.
- **Secondary syphilis**
  - 2-6 months after initial infection
  - Nonspecific symptoms - malaise, anorexia, headache, diffuse adenopathy
  - Generalized maculopapular rash - palms, soles, trunk, limbs
  - Condylomata lata (anogenital, broad-based fleshy grey lesions)
  - Serological tests usually positive.
- **Tertiary syphilis**
  - May involve any organ system
  - Gumma of vulva
  - Neurological – tabes dorsalis, general paresis
  - Cardiovascular – aortic aneurysm, dilated aortic root.
- **Investigations**
  1. Dark ground examination of specimens from cutaneous lesions
  2. Immunofluorescent technique now available for dried lesions
  3. Serological tests
     a. FTA - fluorescent treponemal antibody absorption test
     b. VDRL - flocculation test
     c. Wassermann - complement fixation test.
- **Treatment**
  1. Primary, secondary, latent syphilis of <1 year duration
     - Benzathine penicillin G 2.4 million units IM
     - Treat partners
     - Tetracycline or erythromycin – alternative for penicillin-sensitive women. Tetracycline is contraindicated in pregnancy.
  2. Latent syphilis >1 year duration
     - Benzathine penicillin G 2.4 million units IM once per week x 3 weeks.

CHANCROID (SOFT SORE)

- **Causative Organism –** Haemophilus ducreyi.

- **Clinical Features**
  1. Vesicopustules on the perineum, labia majora, vagina or cervix
  2. Painful tender genital ulcers, sharply demarcated without induration
  3. Heavy foul smelling discharge, which is contagious.
- **Treatment**
  1. Cotrimoxazole – single dose oral
  2. Erythromycin – 500 mg qid for 10 days
  3. Tetracycline – 500 mg qid for 14 days
  4. Ceftriaxone – 250 mg IM single injection
  5. Spectinomycin 2 g IM single injection
  6. Ciprofloxacin 500 mg bd for 3 days
  7. Contact tracing and sexual partner notification
  8. Buboes must be aspirated.

LYMPHOGRANULOMA VENEREUM

- **Causative Organism –** Chlamydia trachomatis.
- **Clinical Features**
  1. Vesicopustules associated with inguinal lymphadenopathy
  2. Ulceration of the inguinal nodes and vulva
  3. Due to secondary infection, there is associated severe pain and movements such as walking or bending are unbearable
  4. Inguinal bubo – bilateral, hard cutaneous induration, reddish blue in color
  5. Anorectal edema causes painful defecation and bleeding per rectum
  6. Rectal strictures
  7. Vaginal stenosis
  8. Systemic symptoms (fever, chills, headache, arthralgia, abdominal cramps) are seen in the later phase
- **Investigations**
  1. Frei's test
  2. ELISA
  3. Complement fixation
  4. Microimmunofluorescence
  5. Culture
  6. Detection of inclusion bodies in the smear from urethra and vaginal ulcer.
- **Treatment**
  1. Tetracycline – 2 g daily for 2-4 weeks and repeated after 1 week
  2. Doxycycline – 100 mg bd for 14 days
  3. Erythromycin – preferred during pregnancy – 500 mg qid for 14 days
  4. Amoxicillin – 500 mg tid for 7 days
  5. Azithromycin – 1 g single dose.

GENITAL HERPES

- **Causative Organism** – *Herpes simplex virus, serotypes 1 and 2 (HSV 2 being more frequent)*
- **Clinical Features**

  Primar – when the infection is acquired

  Characterised by multiple vesicles on vulva, vagina and periurethral area which rupture to form painful erosions causing pain, dysuria and dyspareunia.

  Rec rrent – due to reactivation of HSV, is milder and may be asymptomatic. Frequently affects perineum.
- **Investigations**
  1. Tzanck smear shows multinucleate giant cells
  2. Histopathology
  3. Serology (ELISA, Western blot)
  4. Culture.
- **Treatment**
  1. For primary infection – Acyclovir 400 mg oral tid for 7-10 days.
  2. For recurrences >6 /year – Acyclovir 400 mg oral bd for 6 months.
  3. Severe infection – IV acyclovir 5-10 mg/kg tid for 2-7 days followed by oral drug for a total of 10 days

HIV INFECTION

- HIV is a human retrovirus that causes the acquired immunodeficiency syndrome (AIDS).

Infection can lead to:

- Asymptomatic infection;
- Symptomatic HIV infection;
- AIDS.

- **Transmission**

HIV is transmitted in human body fluids by three major routes:

(1) Sexual intercourse through vaginal, rectal, or penile tissues;
(2) Direct injection with HIV-contaminated drugs, needles, syringes, blood or blood products; and
(3) From HIV infected mother to fetus in utero, through intrapartum inoculation from mother to infant or during breast feeding.

- **Symptoms and signs**
  - Asymptomatic infections by definition are only detected by screening.
  - Symptomatic HIV infection may present with non-specific weight loss, fatigue, lymphadenopathy, diarrhoea and night sweats.
  - AIDS is determined by the onset of certain opportunistic infections or AIDS-related malignancies. Examples include tuberculosis, Pneumocystis carinii pneumonia, cryptococcal infection, cryptosporidiosis, Kaposi's sarcoma and lymphoma.
- **Investigations**
  - Detection of anti-HIV antibodies in the serum by ELISA.
  - Western blots can be performed to investigate discrepant results in reference centres.
  - Plasma viral load gives an estimation of the amount of HIV virus replication.
  - The immune status of the patient is measured by sequential CD4 counts.

➢ **Treatment**

Patients should take three active HIV drugs as highly active antiretroviral therapy (HAART). Classes of drugs include nucleoside analogues (NRTIs), nucleotide analogues (NtRTIs), nonnucleoside analogues (NNRTIs), protease inhibitors (PIs) and fusion inhibitors.

- ➢ NRTIs include zidovudine (AZT), lamivudine (3TC), didanosine (ddI), stavudine (d4T), zalcitabine (ddc), abacavir.
- ➢ NtRTIs include tenofovir.
- ➢ PIs include nelfinavir, saquinavir, lopinavir, ritonavir, indinavir, amprenavir, atazanavir.

➢ **Interventions to Reduce Mother-to-Child Transmission**

1. HIV testing in pregnancy – early detection and treatment
2. Antenatal care – treat STD's and other co-infections
3. Antiretroviral agents – prophylaxis, intrapartum and postpartum regimens.
4. Obstetric interventions
   - Avoid amniotomy
   - Avoid procedures – forceps/vacuum extractor, scalp electrode, scalp blood sampling
   - Restrict episiotomy
   - Elective cesarean section
   - Remember infection prevention practices
   - Newborn feeding – Up to 20% of infants breastfed by HIV-infected mothers are at risk of acquiring HIV. Avoid breastfeeding if replacement feeding is feasible.

➢ **Universal precautions against HIV infection**

1. **Hand hygiene:** Wash hands before and after significant contact with any patient and after activities that may cause contamination.
2. **Gloves:** Wear gloves whenever there is a risk of exposure to blood or body substances, and should change after contact with each patient.
3. **Personal protective equipment:** Eyewear and face shields must be worn during procedures. Impermeable gowns and plastic aprons should be worn to protect clothing and skin from contamination with blood and body substances. Footwear should be enclosed to protect against injury or contact with sharp objects.
4. **Needlestick or sharps injury prevention:** Sharps and clinical waste should be handled carefully at all times.

SYNDROMIC CASE MANAGEMENT OF STD (Now termed as STI's – Sexually Transmitted Infections)

- ➢ STD syndromes are a way to group together infections which cause similar signs and symptoms.
- ➢ Simple flow charts have been developed to help health careworkers use the syndromic approach to make a diagnosis.
- ➢ The most common of the curable STDs are syphilis, gonorrhea, chlamydia, chancroid, trichomoniasis and candidiasis.
- ➢ **Main features /Methods**
  - Classify main causative agents by the clinical syndromes to which they give rise
  - Use flow charts to identify causes of a given syndrome
  - Treat patient for all the important causes of the syndrome
  - Counselling directed towards promotion of safe sexual practices
  - Ensure that partners are treated.

- **Supplementary elements**
  - Patients educated on treatment compliance
  - Patients educated on risk reduction
  - Condoms provided.
- **Benefits of syndromic management**
  - Immediate treatment
  - Effectiveness
  - Ease of use
  - Cost effective.
- Many STD diagnostic tests are expensive. Some tests are also difficult to perform, require special laboratory training or use sophisticated equipment. Syndromic approach does not require the use of these tests which often are not available.
- **Disadvantages of syndromic treatment**
  - Overtreatment in some patients
  - Does not cover asymptomatic infection.

PELVIC ABSCESS

- **Definition:** It is an abscess of the cellular tissues of the pelvis and in the pouch of Douglas.
- **Etiology:**

  It may follow acute salpingitis, aute pelvic peritonitis and cellulitis, rupture of pyosalphinx or appendiceal abscess, infection of the cervix or any other pyogenic bacterial infection.
- **Signs and Symptoms:**
  1. Systemic features of toxicity – fever, malaise, anorexia, nausea, vomiting, pyrexia.
  2. Local effects – pain, deep tenderness in one or both lower quadrants, diarrhea, tenesmus, mucous discharge per rectum, urinary frequency, dysuria, vaginal bleeding or discharge.
  3. Rectal or vaginal examination – may reveal tenderness of the pelvic peritoneum and bulging of the anterior rectal wall.
- **Differential diagnosis**
  1. Pelvic inflammatory disease
  2. Appendicitis
  3. Diverticular disease
  4. Generalised peritonitis – e.g., from a perforated peptic ulcer
  5. Sepsis following termination of pregnancy or miscarriage.
- **Management**
  1. Management is usually by drainage of the abscess along with antibiotic treatment. Antibiotics used alone are occasionally effective for very early, small abscesses.
  2. Antibiotic choice is guided by the likely cause, but usually needs to be broad spectrum until the pathogens are determined.
  3. Procedures used for drainage of the abscess include:
     a) Ultrasound guided aspiration and drainage.
     b) CT guided aspiration and drainage.
     c) Endoscopic ultrasound guided drainage (EUS guided drainage).
     d) Laparotomy or laparoscopy with drainage of abscess may be required in some cases.

Condition	Etiology	Symptoms	Diagnosis	Treatment
Bacterial vaginosis	*Overgrowth of several bacterial species in the vagina*	Non-pruritic vaginal discharge with a "fishy" odor, but 50% of women are asymptomatic.	Positive KOH "whiff" test, pH > 4.5, >20% clue cells on saline wet-mount, gray, homogenous discharge (need 3 of 4 clinical criteria).	Oral/intravaginal metronidazole or clindamycin
Candidiasis	*Candida albicans*	Intense pruritus and vulvovaginal erythema.	KOH wet-mount to see the presence of branched and budding hyphae. Culture in Sabouraud medium may be indicated for selected cases.	Topical clotrimazole or oral fluconazole
Trichomoniasis	*Trichomonas vaginalis*	Profuse malodorous vaginal discharge, postcoital bleeding, vulvovaginal erythema.	Trichomonads seen on saline wet-mount are pathognomonic. Other features include an abundance of leukocytes and pH > 4.5.	Oral (not vaginal) metronidazole.
Chlamydial cervicitis	*Chlamydia trachomatis*	Purulent or mucoid discharge, postcoital bleeding and vaginitis.	DNA probe test obtained vaginally (preferred) or by urine sample, or enyzme-inked immunosorbent assay.	Oral azithromycin or doxycycline.
Gonococcal cervicitis/ vaginitis	*Neisseria gonorrhoeae*	Profuse, odorless, non-irritating, creamy white or yellow vaginal discharge, but may also be asymptomatic. 5% exhibit disseminated gonorrhea infection with chills, fever, malaise, asymmetric polyarthralgias, and painful skin lesions.	A positive culture on selective media such as modified Thayer-Martin agar.	Oral ciprofloxacin
Genital herpes	*Herpes simplex virus (HSV) type 1 (15%) or type 2 (85%).*	First-episode primary HSV infection is characterized by systemic symptoms, including malaise and fever.	Usually clinical inspection is sufficient, but viral isolation by tissue culture is also very reliable.	Oral acyclovir or valacyclovir

		However, genital herpes is a recurrent infection with periods of active infection separated by periods of latency.		
Syphilis	*Treponema pallidum*	Primary infection is marked by a painless, solitary ulcer at the site of acquisition. Secondary symptoms include a facial-sparing rash with involvement of the palms and soles. The classic skin lesion of late syphilis is the solitary gumma nodule.	Dark-field examination of scrapings from a lesion and/or serologic screening (rapid plasma reagin test).	Intramuscular benzathine penicillin.
Genital warts (condyloma acuminatum)	*Human papilloma virus (HPV)*	Uncomplicated cases are asymptomatic.	Usually clinical inspection is sufficient, but colposcopy and/or biopsy may be required.	Cryotherapy, local cytotoxic agents (trichloroacetic acid, podofilox).

PREVIOUS EXAMINATION QUESTIONS

Short Essays	1. Syndromic management of sexually transmitted diseases principles, method and advantages. 2. Complications of gonorrhea in a female patient. 3. Mention laboratory tests of HIV. 4. Diagnosis and treatment of lymphogranuloma venereum. 5. Acute gonorrhea. 6. Prevention of parent to child transmission of HIV. 7. Pelvic abscess.
Short Answers	1. Advice to HIV +ve women. 2. Mention laboratory test of HIV. 3. Genital herpes. 4. Diagnosis and treatment of lymphogranuloma venereum. 5. Chlamydia trachomatis. 6. Pelvic abscess causes and treatment. 7. As a doctor how will you protect yourself from HIV infection when you treat HIV +ve case?

Tuberculosis of the Genital Tract

CHAPTER 12

- **Primary Source –** Lungs 50%, lymphnodes 40%, bones 5%, bladder 5%.
- **Mode of Spread –** Blood (also by sexual and direct spread in children while playing, in 1-2 % cases).
- **Pathology**
  1. Fallopian tube
     - Tuberculous endosalpingitis – tube enlarged, dilated, tortous and frequent attacks due to flow of exudates from tubular ends
     - Tuberculous exosalpingitis – peritoneum is studded by tubercles and ampulla is dilated
     - Interstitial tuberculous salpingitis – tube is thickened
  2. Ovaries – normal or surface studded with tubercles
  3. Uterus – giant cells in endometrium or pyometra → adhesions → Asherman's syndrome → amenorrhea
  4. Cervix – 1-2 % cases present as ulcers
  5. Vulva and vagina – ulcers with undermined edges.
- **Clinical Features**
  1. Infertility – either due to tubal block, impaired ciliary motility or involvement of ovary
  2. Menstrual disorders – 50% normal cycles, 40% menorrhagia and 10% oligomenorrhea and secondary amenorrhea (Ashermann's syndrome)
  3. Pain – due to peritonitis or abscess
  4. Vaginal discharge – blood stained, post coital
  5. Mass per abdomen – frozen pelvis (pelvic masses matted and fixed to uterus)
  6. Fistula formation.
- **Diagnosis**
  1. D and C – premenstrual period when tubercles are present in superficial layers and culture in LJ, BACTEC and HPE
  2. Diagnostic laparoscopy – tubercles on peritoneal surface
  3. USG – pelvic mass due to encysted ascites or formation of tubo-ovarian adhesions
  4. Hystersalpingography (contraindicated)– if done shows lead pipe appearance,beaded appearance, calcification, corneal block, tobacco pouch appearance

5. Search for primary foci – chest X-ray, sputum AFB, urine culture, X-ray bones
6. Latest techniques – PCR, ELISA.

➤ **Treatment**

A. **Medical Line**

1. Anti-TB drugs – The modern regimen consists of:
   a. Isoniazid, rifampicin and pyrazinamide for 2 months
   b. Isoniazid and rifampicin biweekly for another 5-6 months.
2. Corticosteroids – may be needed in an ill patient.
3. Resistant cases associated with HIV need extended treatment for a year.
4. Newer drugs:
   a. Quinolones
   b. Clofazimine
   c. Ampicillin
   d. Amikacin.

B. **Surgical Line**

1. Indications:
   a. Progression of the disease
   b. Persistent active lesion
   c. Persistence of large inflammatory masses (pyosalpinx, pyometra)
   d. Persistence of symptoms (pain, menorrhagia)
   e. Persistence of fistula.
2. Contraindications:
   a. Active lesions elsewhere in the body
   b. Plastic adhesion of bowels.
3. Types of surgery:
   a. Total hysterectomy with removal of ovaries and fallopian tubes - rare
   b. Vulvectomy – in cases of hypertrophied vulva
   c. Tuboplasty – contraindicated.

C. **Follow-up**

1. Patient should be followed up for at least 5 years, as reactivation of the lesion during this period has been reported
2. Yearly (or when indicated earlier) curettage to check for any reactivation.

PREVIOUS EXAMINATION QUESTIONS	
Long Essays	1. Discuss etiology, pathogenesis, diagnosis and management of genital tuberculosis in female.
Short Essays	1. Types of TB salpingitis. 2. Medical treatment for pelvic tuberculosis. 3. Treatment of genital tuberculosis. 4. Treatment of TB pyosalpinx. 5. Medical management of genital tuberculosis.
Short Answers	1. Menstrual problems in a patient with genital tuberculosis. 2. USG finding in genital tuberculosis. 3. Diagnosis and treatment of tuberculosis endometrium. 4. Ashermann's syndrome.

Diseases of the Urinary System

CHAPTER 13

RETENTION OF URINE

➢ **Causes (Acute)**

1. **Post-operative –** Common after vaginal and perineal operations
   a. Post-operative edema may cause obstruction to the flow of urine
   b. Pain from pelvic region may lead to reflex spasm of the bladder sphincter
   c. Radical operations like Wertheim's hysterectomy involve extensive dissection causing denervation of the bladder
   d. Spinal and epidural anesthesia account for retention in the first 12-24 hours of the postoperative period.
2. **Puerperal retention –** After delivery, the patient is often unable to appreciate the filling of the bladder due to bruising of the vagina and painful perineal wounds.
3. **Space-occupying lesion in the pelvis –** Obstruction of urethra or bladder neck
   a. Retroverted gravid uterus (14 weeks of gestation)
   b. Deeply engaged presenting part during labor
   c. Hematocolpos in adolescent girls
   d. Hematocele complicating an ectopic gestation
   e. Cervical myomas
   f. Posterior uterine wall myoma impacted in pouch of Douglas
   g. Ovarian neoplasm impacted in the pelvis.
4. **Obstructive conditions**
   a. Cicatricial stenosis – following surgery on the bladder neck for a fistula or in the urethra for a caruncle
   b. Inflammatory stenosis – following gonorrhoea
   c. Sling operations for stress incontinence may occlude the bladder
   d. Cancer of the cervix, vagina, bladder or urethra may lead to extensive tissue infiltration and obstruction
5. **Neurological**
   a. Spinal cord lesions
   b. Disseminated sclerosis
   c. Tabes dorsalis
   d. Denervation of the bladder.

- **Causes (Chronic)** - bladder neck obstruction.
- **Treatment**

1. Acute retention
   a. Catheterization with autoclaved soft rubber catheter
   b. If patient fails to pass urine normally after 8 hours, a self-retaining catheter is introduced for continuous drainage for at least 24-48 hours.
2. Chronic retention – Self-retaining catheter is introduced. The number of days it is to be kept depends on the regaining of the bladder tone. This is determined by the amount of residual urine (which should preferably be below 50 ml and never be allowed to exceed 100 ml).

URINARY INCONTINENCE

- **Definition –** Objectively demonstrable involuntary loss of urine so as to cause hygienic and/ or social inconvenience for day-to-day activity.
- **Types**

1. Stress incontinence - involuntary loss is associated with stressful activity like coughing, sneezing, straining or other physical activity
2. Urge incontinence - involuntary loss follows a strong desire and need to void
3. Unconscious incontinence - no awareness of urinary leak.

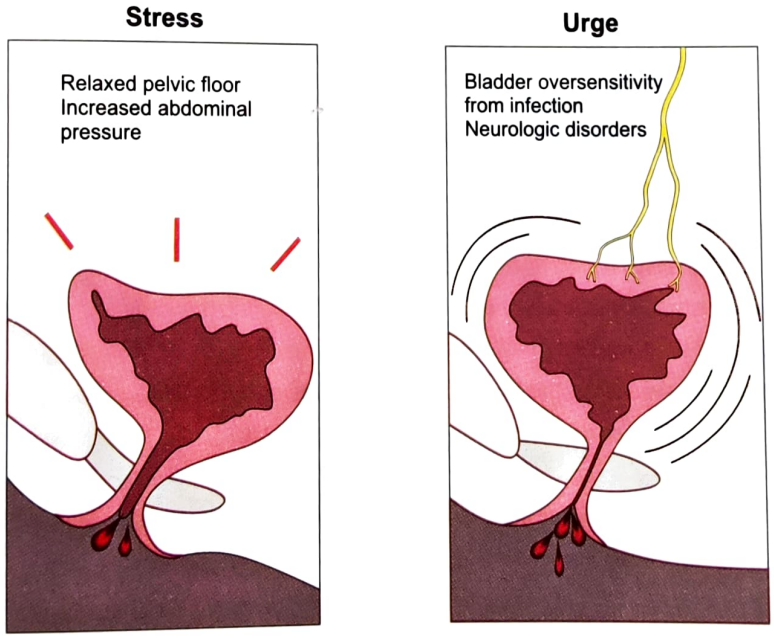


Fig. 13.1: Types of urinary incontinence.

➢ **Pathophysiology**

- Continence and micturition involve a balance between urethral closure and detrusor muscle activity. Urethral pressure normally exceeds bladder pressure, resulting in urine remaining in the bladder. The proximal urethra and bladder are normally both within the pelvis. Intra-abdominal pressure increases are transmitted to both urethra and bladder equally, leaving the pressure differential unchanged, resulting in continence.
- In stress incontinence, rise in bladder pressure because of intra-abdominal pressure increases (e.g., coughing and sneezing) are not transmitted to the proximal urethra, because it is no longer a pelvic structure owing to loss of support from pelvic relaxation.
- In urge incontinence, involuntary rises in bladder pressure occur from idiopathic detrusor contractions that cannot be voluntarily suppressed.

➢ **Clinical Features**

1. History
   a. Stress incontinence – Patient complains of passage of a single spurt of urine at the height of physical exertion, like sneezing or coughing.
   b. Urge incontinence – Patient complains of a strong desire to void, which if not complied with immediately, leads to a more prolonged involuntary passage.
2. Physical Examination
   a. Clinical examination, including pelvic and speculum examination and a thorough neurological assessment should be done.
   b. Assessment of anatomic defects of pelvic supports and tone of levator muscles.
   c. Assessment of vaginal wall prolapse and senile vaginal changes.

➢ **Investigations**

1. Stress test

- Patient voids urine, then catheterize and collect sample of residual urine.
- The urine sample is sent for culture.
- 250 ml warm saline is filled into bladder.
- Patient is made to squat on a absorbent pad placed on the floor and asked to cough.
- Pad is weighed, if weight >2 g then it is stress incontinence.

2. Cotton swab stick test

- A cotton swab stick is placed in the urethra (dipped in xylocaine).
- Patient is asked to strain and cough.
- Initially it will be parallel to the floor and normally raises by 10-15 degrees above the horizontal.
- Raises more than 20 degree in case of stress incontinence.

3. Marshall test

- Vagina in the region of bladder neck is infiltrated with local anesthesia.
- The area is elevated with an open Allis clamp.
- Failure to demonstrate leakage of urine on coughing indicates a positive result.

4. Bonney's test

- 2 fingers are placed in the vagina at urethrovesical junction on either side of urethra and bladder, neck region is elevated.

- On straining or coughing, absence of leakage indicates a positive test.

5. **Urethroscopy**
6. **Lateral cystourethrogram**
7. **Ultrasound –** to assess the bladder neck and volume of residual urine.
8. **Urodynamic studies –** Urodynamics are a means of evaluating the pressure-flow relationship between the bladder and the urethra for the purpose of defining the functional status of the lower urinary tract. It aids in the diagnosis of urinary incontinence based on pathophysiology. It includes MCU, cystometry, urethrocystometry, uroflowmetry, urethral pressure and urethral closure pressure profiles.

   Cystometry: Involves the measurement of pressure/volume relationship of the bladder during filling and voiding and is the most useful test of bladder function. In genuine stress incontinence, there will be leakage on coughing without rise in the detrusor pressure. In detrusor instability, spontaneous detrusor contractions are noted while the woman is attempting to inhibit micturition.

   Uroflometry: It's a simple non-invasive test done in the outpatient setting. The normal flow rate is 15 ml per second with the voided volume above 150 ml. A low peak flow rate and a prolonged voiding time suggests a voiding disorder.

➢ **Treatment**

1. **Conservative Treatment** – It should be the first line of treatment in younger women. It is also applied to elderly and frail women unfit for surgery and during the 6 months after delivery.
   a. Physiotherapy – pelvic floor exercises for 4 months.
   b. Drugs – alpha-adrenergic agents, estrogen cream.
   c. Intraurethral and vaginal devices – ring pessary, contiform.
   d. Electric stimulation – in case of denervation of the pudendal nerve.
   e. Artificial urinary sphincter (AUS) – AUS 800 model is used.
2. **Surgical Repair**
   a. Vaginal operations
      1) Anterior colporrhaphy
      2) Kelly's repair – cystourethroplasty usually combined with cystocele repair. Success rate is low
      3) Pacey's repair.
   b. Abdominal operations (Retropubic bladder neck suspension operations)
      1) Marshall-Marchetti-Krantz operation – vaginal wall is sutured to the pectineal ligament
      2) Burch colposuspension – vaginal wall is sutured to the periosteum of the back of pubic bone.
   c. Combined operations – needle suspension techniques which are outdated
      1) Pereyra operation
      2) Razz and Stamey modifications.
   d. Sling procedures

      Multiple varieties using synthetic substances (nylon, prolene, Teflon, mersilene mesh, tension-free vaginal tapes) or natural tissues (e.g., round ligament or external oblique aponeurosis). Tension-free transvaginal tape (TVT) or transobturator tape (TOT) are minimally invasive suburethral sling procedures that are rapidly becoming the "gold standard."

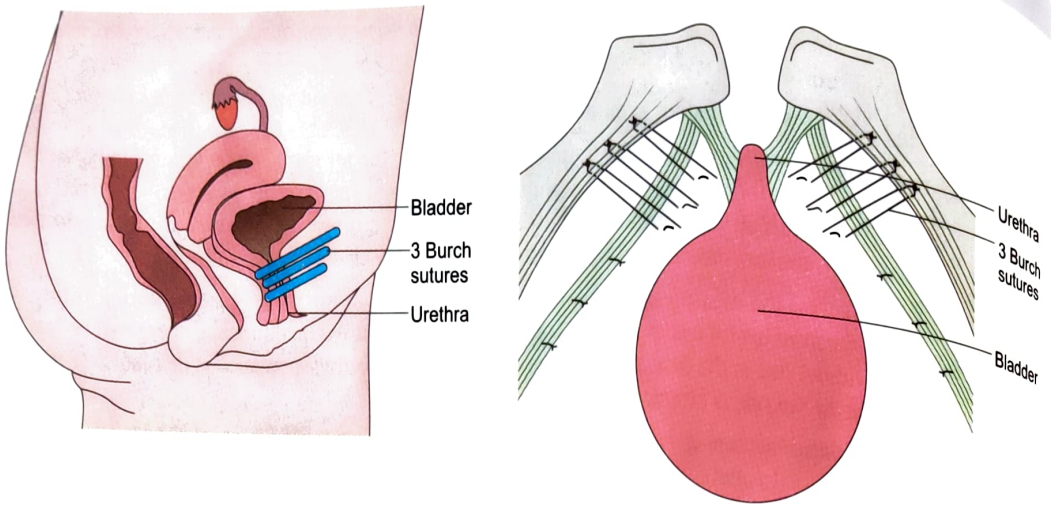


Fig. 13.2: Burch colposuspension

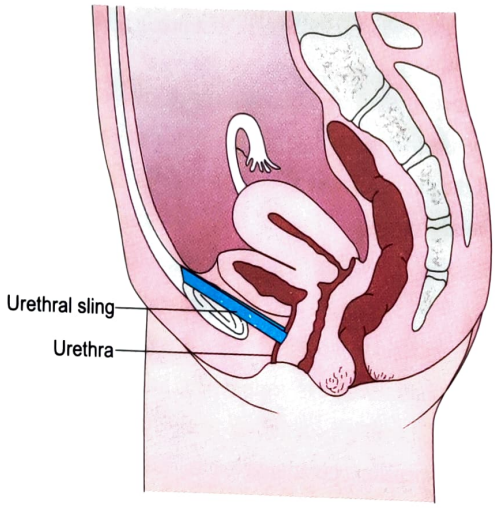


Fig. 13.3: TVT sling surgery

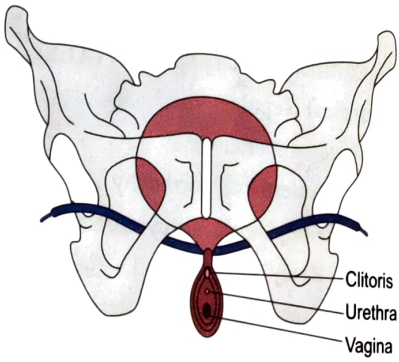


Fig. 13.4: Transobturator tape.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. Define, enumerate causes and discuss the management of urinary incontinence. 2. Management of stress urinary incontinence. 3. Investigations and treatment of stress urinary incontinence. 4. Define urodynamics of stress incontinence. How would you evaluate and treat a patient with genuine stress urinary incontinence?
Short Essays	1. Gynecological causes of urinary retention. 2. What are the causes of incontinence of urine? How will you manage stress incontinence? 3. Stress incontinence. 4. Management of stress urinary incontinence.
Short Answers	1. Asymptomatic bacteruria. 2. Investigations for stress incontinence. 3. Kelly's operation indication and principle. 4. Causes of urinary retention in gynecology. 5. Enumerate causes of acute retention of urine. 6. What are the causes for retention of urine in gynecological practice? 7. Describe the causes of retention of urine in 60 year old woman. 8. Causes of obstructive uropathy in gynecology. 9. Urge incontinence. 10. Diagnosis of stress incontinence.

Genital Fistulae

CHAPTER 14

GENITAL FISTULAE

- **Definition –** Abnormal epithelialized communication tracts between the genital tracts and urinary or alimentary tracts or both.
- **Classification**
  1. Bladder
     a. Vesico-vaginal (commonest)
     b. Vesico urethro-vaginal
     c. Vesico uterine
     d. Vesico cervical
  2. Urethra
     Urethro-vaginal
  3. Ureter
     a. Uretero-vaginal
     b. Uretero-uterine
     c. Uretero-cervical.
- **Etiology**
  1. Obstetrical injury
     a. Prolonged obstructed labor
     b. Difficult instrumental or manipulative delivery (e.g. forceps)
     c. Injury to bladder during caesarean section
     d. Perforators and speckles of bone during craniotomy operation
     e. Symphysiotomy
     f. Rupture of the uterus.
  2. Operation injury
     a. Pan hysterectomy
     b. Wertheim's radical abdominal operation for cancer cervix

c. Total abdominal hysterectomy

d. Vaginal hysterectomy

e. Internal iliac ligation

f. Criminal abortion

g. Pelvic surgery for endometriosis, PID, cervical fibroid.

3. Laparoscopic injuries – direct trocar injuries to the urinary bladder, ureter, bowel, sigmoid colon and rectum.
4. Radiation – bladder cannot tolerate the same dose of irradiation as the cervix.
5. Congenital fistula.
6. Careless catheterisation.

➢ **Clinical Features**

1. Constant dribbling of urine
2. Excoriation of the vagina, vulva, perineum and thighs – due to constant wetness
3. These women are depressed and treated as social outcasts
4. Speculum examination – exposes the fistula and enables clinical assessment of its size and location
5. Bimanual examination – reveals information about its fixity and extent of scarring of the surrounding tissue
6. Positive methylene blue test – confirms the diagnosis.

➢ **Investigations**

1. Complete blood count
2. Urinalysis
3. Renal function test
4. Urine culture – mandatory before surgery; infection should be treated
5. Sonography of kidney, ureter and bladder
6. Intravenous pyelography (IVP)
7. Cystoscopy with indigocarmine excretion test enables visualization of the dye from each ureteric orifice individually
8. Ureteric catheterization
9. Methylene blue test (3 swab test)

a. Procedure

1) Catheter is introduced into the bladder through the urethra

2) Vaginal cavity is packed with 3 sterile swabs – one at the vault, one in the middle and one just above the introitus

3) 50-100 ml of diluted methylene blue dye is injected into the bladder through the catheter.

b. Inference

1) If there is a VVF, the methylene blue dye stains the uppermost swab

2) If the leak is from the urethra, the lowermost swab gets stained (urethrovaginal fistula)
3) If the leak is from the ureter, the swabs do not take up the stain, but get wet with urine (ureterovaginal fistula).

➤ **Treatment**

1. Indwelling catheter and prolonged draining of the bladder
2. Antibiotics
3. Supportive therapy
4. Latzko procedure – vaginal repair. Vaginal epithelium is denuded all around the fistulous edge, edge is freshened and the wide raw surfaces are approximated with rows of absorbable sutures
5. Chassar Moir technique – Vagina and bladder are widely separated all around by the flap splitting method. Bladder and vagina are sutured separately in 2 layers
6. If one attempt fails to heal the fistula, second vaginal repair can be undertaken after a period of 3 months
7. Urinary diversion (implantation of the ureters into the sigmoid colon, creating an ileal loop bladder into which the ureters are implanted or a rectal bladder) is considered in case of extensive loss of bladder tissue, previous repetitive failures to close the fistula or radiation fistula which fails to heal
8. Post-operative management
   a. Continuous bladder drainage for 14 days
   b. Antibiotics to adequately treat urine infection
   c. No vaginal or speculum examination for 3 months
   d. No sexual intercourse for 3 months
   e. Caesarean section is indicated following successful fistula repair.

RECTOVAGINAL FISTULA

➤ **Definition** – A rectovaginal fistula is an abnormal connection between the rectum and vagina.

➤ **Causes:**

1. Injuries during childbirth – injuries during delivery are the most common cause
2. Crohn's disease – the second most common cause of rectovaginal fistulae
3. Cancer or radiation treatment in the pelvic area
4. Surgery involving the vagina, perineum, rectum or anus
5. Other causes – rarely, a rectovaginal fistula may be caused by infections in the anus or rectum

➤ **Clinical features:**

1. Passage of gas, stool or pus from vagina
2. Foul smelling vaginal discharge
3. Recurrent vaginal or urinary tract infections

4. Irritation or pain in the vulva, vagina and the perineum
5. Pain during sexual intercourse.

➢ **Diagnosis**

1. Contrast tests – a vaginogram or a barium enema.
2. Blue dye test – This test involves placing a tampon into the vagina, then injecting blue dye into the rectum. Blue staining on the tampon shows the presence of a fistula.
3. Computerized tomography (CT) scan.
4. Magnetic resonance imaging (MRI).
5. Anorectal ultrasound.
6. Anorectal manometry – helps in planning to repair the fistula.

➢ **Treatment**

1. Treatment for the fistula depends on its cause, size, location and effect on surrounding tissues.
2. Antibiotics if there is infection. Infliximab to reduce inflammation in Crohn's disease.
3. Sometimes fistulas heal on their own, but most people need surgery. A waiting period of three to six months before surgery to ensure the surrounding tissue is healthy.
4. Surgical options include sewing an anal fistula plug or patch of biologic tissue or tissue graft into the fistula to allow the tissue to grow into the patch and heal the fistula. Colostomy may be needed in recurrent cases.

PREVIOUS EXAMINATION QUESTIONS	
Long Essays	1. Enumerate the causes, investigations, diagnosis and management of vesicovaginal fistula. 2. Postoperative care-VVF repair.
Short Essays	1. Cause and management of genitourinary fistula. 2. Types and etiology of vesicovaginal fistula. 3. Ureteric injuries in gynecological surgeries. 4. Urinary fistulae. 5. Diagnosis of genitourinary fistula. 6. Postoperative care for vesicovaginal fistula repair. 7. Causes of gynecological urinary fistulae.
Short Answers	1. Causes of genitourinary fistulae. 2. Causes of gynecologic urinary fistulae. 3. Vesicovaginal fistula. 4. Three swab test in VVF and its inference. 5. Treatment of rectovaginal fistula. 6. Postoperative care and advice after repair of vesicovaginal fistula. 7. Causes for rectovaginal fistula.

Pathology of Conception

CHAPTER 15

DEFINITION

A. Sterility – absolute inability to conceive
B. Infertility – failure to conceive within 1 year of regular unprotected intercourse
C. Primary infertility – patient who has never conceived
D. Secondary infertility – patient fails to conceive after achieving a previous conception.

MALE INFERTILITY

➤ **Causes**

1. Defective spermatogenesis
   a. Undescended testis
   b. Orchitis (mumps)
   c. Genetic (47 XXY)
   d. Testicular toxins (drugs, radiation)
   e. Endocrinal (thyroid dysfunction, gonadotrophin deficiency)
   f. Varicocele
   g. Primary testicular failure.
2. Obstruction of efferent duct
   a. Congenital
      1) Absence of vas deferens (cystic fibrosis)
      2) Young's syndrome
   b. Acquired
      1) Infection (TB, gonorrhea)
      2) Surgical trauma (herniorrhaphy, vasectomy).
3. Failure to deposit sperm high in vagina
   a. Impotency
   b. Ejaculatory failure
   c. Retrograde ejaculation

d. Hypospadias
e. Bladder neck surgery
f. Drug related
g. Psychosexual.

4. Defect in sperm and seminal fluid
   a. Kartagener's syndrome (immotile sperm)
   b. Asthenozoospermia
   c. Teratozoospermia
   d. Low fructose content
   e. Sperm antibodies.

➢ **Investigations**

1. History – Age, occupation, previous children, duration of infertility, any contraception practiced and for how long, coital frequency and timing, medical and surgical history, habits (smoking, alcohol, drugs), coital difficulties.
2. General examination
   a. Height – increased in Kallmann and Klinefelter's syndrome
   b. Weight and obesity – may be a hormonal defect
   c. Secondary sex characteristics – abnormal in Klinefelter's syndrome
   d. Thyroid enlargement
   e. Gynecomastia
   f. Blood pressure.
3. Local examination - penis, scrotum, surgical scar.
4. Special investigations
   a. Semen analysis
   b. Testicular biopsy
   c. FSH level
   d. Chromosomal study
   e. Immunological disorders
   f. Ultrasound.
5. Semen analysis – This is the most important part of the male investigation. The best specimen is one obtained by masturbation in the vicinity of the laboratory, since this guarantees its freshness. If this is objectionable to the man, coitus interruptus into a wide-necked bottle may be employed. Another method is the post-coital test (described later). The best specimen will be produced if a short period of abstinence of 3-5 days is observed. The specimen should be examined within 2 hours of production.
   a. Normal features
      1) Coagulate soon after ejaculation due to enzyme in the seminal vesicle but liquefy in 30 minutes because of prostatic enzyme
      2) Color – greyish white
      3) Consistency – viscous

4) Volume – 3-5 ml (average 3.5 ml)
5) pH – 8
6) Fructose – present
7) Pus cells – absent
8) Sperm count – 60-120 million per ml (average 100 million)
9) Sperm motility – 80-90% (average 80%); 10 motile sperms per high field
10) Sperm morphology - 80% or more normal (average 80%)

b. Terminology
1) Aspermia – no semen
2) Azoospermia – no sperm in semen
3) Asthenospermia – no motile sperm or diminished motility
4) Necrospermia – dead sperms
5) Teratospermia – abnormal morphology of sperms
6) Post-coital test (Sims' or Huhner's test) – Couple is advised intercourse close to ovulation time, preferably in the early hours of the morning. The woman presents herself at the clinic within 2 hours after the intercourse. The mucus is aspirated from the cervical canal and spread over a glass slide. Another smear made from the posterior fornix serves as a control. Normally 10-50 motile sperms are seen per high power field in cervical mucus. If there are less than 10 sperms, proper semen analysis should be undertaken.

➢ **Treatment**

1. Education – sexual counselling (frequency, timing, position, etc).
2. Substance abuse – avoidance of tobacco, alcohol, drugs, etc.
3. Reduce heat around the scrotum – loose cotton underwear, control obesity, avoid hot baths, avoid strenuous activities, etc.
4. Correct endocrine disorders – thyroid disorders, diabetes, etc.
5. Surgery – correction of varicocele, undescended testis, etc.
6. Antibiotics – doxycycline for treatment of infections (orchitis,prostatitis, etc).
7. Hormone therapy – hCG, hMG, testosterone, clomiphene, tamoxifen, GnRH, dexamethasone, prednisone, cyclosporin A, etc.
8. Sildenafil (Viagra) – 25-100 mg one hour before intercourse improves erectile function. It is contraindicated in men on antihypertensives.
9. Artificial insemination
   a. Indications
      1) Oligospermia after sperm washout
      2) Impotency
      3) Premature ejaculation
      4) Retrograde ejaculation
      5) Hypospadias
      6) Anti-sperm antibodies in cervical mucus
      7) Unexplained infertility
      8) Donor semen

b. Techniques
   1) Intra-uterine and intra-fallopian done via hysteroscope or blind procedure
   2) Intra-cervical
   3) Pan-cervical
   4) Vaginal
   5) DIPI (direct intra-peritoneal insemination).

FEMALE INFERTILITY

➢ **Causes**

1. Dyspareunia and vaginal causes – fixed retroversion with prolapsed ovaries, inflamed adnexal disease, pelvic endometriosis
2. Infection in the lower genital tract – Neisseria gonorrhoeae, Chlamydia trachomatis, Ureaplasma urealyticum
3. Congenital defects in the genital tract – absent uterus, absent or septate vagina
4. Cervical factors – position of cervix and patency of cervical canal
5. Uterine factors – hypoplasia, malformations, incompetent os
6. Tubal factors – salpingitis, tubal blockage, peri-tubal adhesions
7. Ovarian factors – PCOD, corpus luteal phase defects
8. Chronic ill health – hypothalamic and pituitary disease, hypothyroidism
9. Peritoneal factors – PID.

➢ **Investigations**

1. History – Age, past obstetric history, medical and surgical illnesses, duration of infertility, contraceptive usage.
2. Examination – Height, weight, blood pressure, hirsutism, lymph nodes, thyroid, breasts, abdominal examination, pelvic examination.
3. Tests for ovulation (described later).
4. Tests for tubal patency
   a. Hysterosalpingography – Involves injection of a radioopaque dye through the uterus with fluoroscopic visualization of the uterine cavity and tubal lumina. Optimally done within 10 days of a menstrual cycle. Normal finding shows the cavity of the uterine body triangular in shape with the diameter of the corneal portion approximately 35 mm. The fallopian tubes are 5.6 cm long and free spill of the contrast into the peritoneal cavity should be seen if the tubes are patent.
   b. Sonosalpingography – It is performed using ultrasonography, using an ultrasound medium which contains galactose microparticles and therefore free of the possible risks of radiation.
   c. Laparoscopic chromotubation – During laparoscopy, status of the tubes is inspected by injecting methylene blue into the uterus and shown to pass through the tubes when the cervix is occluded.
   d. Hysteroscopy – involves evaluation of endometrial cavity and fallopian tubes using a hysteroscope.
   e. Transcervical falloscopy.
   f. Ampullary salpingoscopy.
   g. Fimbrial salpingoscopy.

   Complications: Allergic reaction, extreme pain leading to arrythmias, pelvic infection, uterine rupture, tubal rupture, and hemorrhage.

➢ **Treatment**

1. Tubal microsurgery
2. Laparoscopy
   a. Tubal adhesiolysis b. Fimbrioplasty c. Tubal surgery
3. Balloon tuboplasty and cannulation
4. Fallopian tube recanalization- removes fallopian tube blockage using special catheters.
5. IVF (in-vitro fertilization)
6. GIFT (gamete intra-fallopian transfer)
7. Treatment of uterine causes (septum, fibroid, Asherman syndrome, etc.).

TESTS OF OVULATION

➢ **Basal Body Temperature (BBT)**

1. There is biphasic pattern of temperature variation in ovulatory cycle.
2. If pregnancy occurs, rise of temperature sustains along with absence of the period.
3. In anovulatory cycle, there is no rise of temperature throughout the cycle.

➢ **Endometrial Biopsy** – Evidences of secretory activity of the endometrial glands in the second half of the cycle give the diagnosis of ovulation and can predict the functional integrity of the corpus luteum.

➢ **Fern Test**

1. During the mid-cycle, the cervical mucus is obtained by a platinum loop or pipette and spread on a clean glass slide and dried. When seen under low power microscope, it shows characteristic pattern of fern formation.
2. It is due to high sodium chloride and low protein content in the mucus due to high estrogen in the mid-menstrual phase prior to ovulation. After ovulation with increasing progesterone, the ferning disappears completely after 21st day.
3. Thus, the presence of ferning even after 21st day suggests anovulation and its disappearance is presumptive evidence of ovulation.

➢ **Spinnbarkeit (stretchability or elasticity)**

1. During the mid-cycle, the cervical secretion is collected with a pipette and placed over a glass slide. Another glass slide is placed over it.
2. Because of increased elasticity due to high estrogen level during this period, the mucus placed between the slides can withstand stretching up to 10 cm.
3. After ovulation when corpus luteum forms, progesterone is secreted. Under its action, the cervical mucus loses its property of elasticity and while attempting the above procedure, the mucus fractures when put under tension much earlier.
4. This loss of elasticity after its presence is indirect evidence of ovulation.

➢ **Insler Scoring System**

1. It takes into account state of the external os and various cervical mucus properties such as the amount, spinnbarkeit, ferning, viscosity and cellularity.
2. The maximum score is 15 and a score of less than 10 is considered unfavorable.

➢ **Ultrasound** – daily ultrasound visualization of the ovaries from 10th -16th day of the menstrual cycle.

➢ **Hormonal Study** – plasma progesterone, prolactin, FSH, LH, thyroid tests.

INDUCTION OF OVULATION

Ovulatory infertility can be treated using the following drugs:

1. **Clomiphene citrate**
   - *Indications* – The most common medication used to induce ovulation. The treatment of choice for women with chronic anovulation but adequate levels of estrogen and eugonadotropic eugonadism.
   - *Mode of action* – Clomiphene is a selective estrogen receptor modulator (SERM). It reduces the negative feedback effect of circulating estrogen, thereby triggering hypothalamic gonadotropin releasing hormone (GnRH) secretion, and increased release of pituitary gonadotropins (FSH/LH) leading to follicular recruitment, selection, and ovulation 5–12 days after the last dose.
   - *Dosage* – The initial dose is 50 mg daily for 5 days beginning on day 3 or 5 of the menstrual cycle. The dose is increased in each cycle in 50 mg increments until ovulation is observed.
2. **Human menopausal gonadotropins (hMGs) or recombinant follicle-stimulating hormone (rFSH)**
   - *Indications* – Human MG is the treatment of choice for women with ovulatory dysfunction and low levels of estrogen and gonadotropins (hypogonadotropic hypogonadism).
   - *Mode of action* – Human MG is a purified preparation of gonadotropins (LH, FSH) extracted from the urine of postmenopausal women. Recombinant FSH is produced from genetically engineered cell lines. Administration promotes follicular growth and maturation.
   - *Dosage* – Dosage is variable and dependent upon ovarian reserve.
3. **Bromocriptine**
   - *Indications* – Bromocriptine is indicated only for women with hyperprolactinemic ovulatory dysfunction caused by prolactin-secreting pituitary adenomas or idiopathic hyperprolactinemia.
   - *Mode of action* – Elevated prolactin levels interfere with the normal menstrual cycle by suppressing pulsatile secretion of GnRH by the hypothalamus. Bromocriptine is a dopamine agonist that inhibits the pituitary secretion of prolactin.
   - *Dosage* – The ideal maintenance dose is 2.5 mg. It may be increased weekly in 1.25 mg increments until normal menstruation is achieved.
4. **Pulsatile GnRH**
   - *Indications* – Pulsatile GnRH therapy (subcutaneous pump) is indicated in patients with hypothalamo-pituitary dysfunction.
   - *Mode of action* – Exogenous pulsatile GnRH stimulates pituitary gonadotropin release and thus ovulation.
   - *Dosage* – GnRH is administered via intravenous (5–10 mg per pulse) or subcutaneous (10–20 mg per pulse) injection.

O arian h perstim lation s ndrome

- Ovarian hyperstimulation syndrome (OHSS) is a rare, iatrogenic complication of ovarian stimulation. It may occur after using injectable hormone medications.
- This syndrome is characterized by ovarian enlargement due to multiple ovarian cysts and an acute fluid shift into the extravascular space.
- Symptoms are set into 3 categories – mild, moderate, and severe.
  Mild symptoms include abdominal bloating and feeling of fullness, nausea, diarrhea, and slight weight gain.
  Moderate symptoms include excessive weight gain, increased abdominal girth, vomiting, diarrhea.
  Severe symptoms are fullness/bloating above the waist, shortness of breath, pleural effusion, urination significantly darker or has ceased, marked abdominal bloating or distention, and lower abdominal pains.

ASSISTED REPRODUCTIVE TECHNIQUES (ART)

➢ **Definition** – Assisted reproductive technology (ART) is the application of laboratory or clinical technology to gametes (human egg or sperm) and/or embryos for the purposes of reproduction.

➢ **Indications:**

1. Mechanical infertility
2. Defective seminal parameters:
   a. Low count, motility or abnormal forms
   b. Low or high volume, high viscosity or abnormal liquefaction
3. Immune infertility
4. Idiopathic infertility.

➢ **Methods:**

1. IVF (In-vitro fertilization)

This technique involves:

- Ovulation induction, for better egg retrieval which could result in a higher pregnancy rate. (controlled ovarian hyperstimulation)
- Ultrasound evaluation of the follicles stimulated during ovulation induction.
- Egg retrieval, via ultrasound-guided needles to suction the eggs out of the follicles.
- Insemination in a dish, with resulting conception of one or more embryos.
- Embryo transfer.

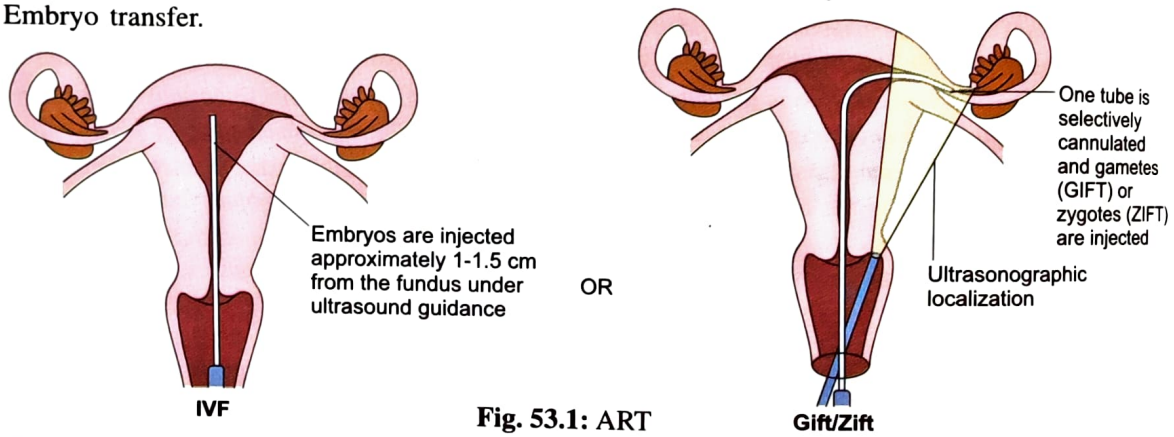

Embryos are injected approximately 1-1.5 cm from the fundus under ultrasound guidance
IVF
OR
One tube is selectively cannulated and gametes (GIFT) or zygotes (ZIFT) are injected
Ultrasonographic localization
Gift/Zift

Fig. 53.1: ART

2. GIFT (Gamete intrafallopian transfer)

In this technique, eggs and sperm are injected into a woman's fallopian tube(s), where fertilization takes place. A woman must have at least one normal fallopian tube in order for GIFT to be suitable. It is used in instances where the fertility problem relates to sperm dysfunction, and where the couple has idiopathic (unknown cause) infertility.

3. ZIFT (Zygote intrafallopian transfer)

In this technique, a fertilized egg via the IVF technique is injected into the fallopian tube, as in GIFT. Conception takes place in the lab. The fertilized egg i.e., the zygote rolls down toward the intrauterine cavity, like in normal fertilization.

4. Artificial insemination

Sperms are injected into the uterus from a vaginal approach. Fertilization still takes place in the fallopian tube. This procedure is indicated in conditions where sperm deposition is a problem e.g., in ejaculatory failure, gross hypospadias, erectile dysfunction, structural deformity of penis due to trauma.

5. ICSI (Intracytoplasmic sperm injection)

In this process, a single sperm is injected into the egg, is used when the sperm count is low or only a small percentage of sperms are healthy.

PREVIOUS EXAMINATION QUESTIONS

Long Essays

1. Describe infertility in female investigations and management.
2. Describe fallopian tube and discuss tubal causes of infertility.
3. Enumerate the causes of primary infertility.
4. How do you manage the case of primary infertility?
5. Enumerate the causes of secondary infertility. How do you manage the case of secondary infertility?
6. Male factor of infertility investigation and management.
7. Discuss the etiology, diagnosis and management of infertility in the female.
8. What is the cause of infertility? Describe how you will investigate a couple with infertility.
9. Enumerate causes of "female infertility". What are the signs, symptoms and tests for ovulation? How will you treat an ovulatory infertility?
10. Mention causes of female infertility. What are the tests for ovulation? And discuss semen analysis.
11. What are the factors to be assessed in the infertility work up of the female partner? How will you assess the tubal factor in infertility?
12. How will you evaluate female factors in infertility?

Short Essays

1. Classify causes of male infertility.
2. Controlled ovarian hyperstimulation.
3. Diagnostic tests for tubal factor in infertility.
4. Intrauterine insemination indication(s), technique.
5. Tubal patency tests and their interpretation.
6. Timing and tests of tubal patency.
7. Assessment of tubal factor in infertility.
8. Non-invasive investigations in sterility of a female patient.
9. Complications of tubal patency tests.
10. Management of tubal factor infertility.
11. Signs, symptoms and diagnosis of ovulation.
12. Investigations for ovulation.
13. Induction of ovulation drugs used and monitoring.
14. Male infertility investigations.
15. Sim's coital test.
16. Post coital test.
17. Anovulation
18. Semen analysis and its interpretation.
19. What is assisted reproduction? Mention the various types used in technology of assisted reproduction.
20. Tubal patency tests.
21. Detection of ovulation.
22. Tests of ovulation.
23. Ovulation induction methods.
24. What are the uses of ultrasound in infertility?

Short Answers	1. Write normal semen analysis report. 2. Intrauterine insemination indication(s) and technique. 3. Investigations for female partner in work-up of infertility. 4. Indications for artificial reproductive technology (ART). 5. Management of hyperprolactinemia. 6. Clomiphene citrate – clinical use. 7. Enumerate ART techniques. 8. Causes of secondary infertility. 9. Anatomy of normal sperm. 10. Mention causes of secondary infertility. 11. Semen analysis. 12. Post-coital test. 13. Normal parameters of semen analysis (as suggested by WHO)/ normal semen analysis report. 14. What is GIFT? What are the indications? 15. Mention tubal patency tests. 16. Sonosalpingography. 17. Tubal recanalization. 18. Fern test. 19. Describe the methods of induction of ovulation. 20. Enumerate drugs used for ovulation induction. 21. Surrogate motherhood.

Birth Control and Medical Termination of Pregnancy

CHAPTER 16

PEARL INDEX

- A method used to determine the failure (pregnancy) rate of any contraceptive technique
- Pregnancy rate = $\frac{\text{No. of pregnancies} \times 1200}{\text{Women - months of use}}$.
- Hence, failure rate = Pregnancy rate per 100 woman years.
- A high Pearl index stands for a high chance of unintentionally getting pregnant; a low value for a low chance.

NATURAL CONTRACEPTION

- **Safe Period/Rhythm Method**

  This is based on the avoidance of sexual intercourse during the fertile period of the cycle.
- **Safe period**

  1. Calendar rhythm 2. Temperature rhythm 3. Mucus rhythm
- **Methods**

  a) Recording of previous menstrual cycles (calendar rhythm) –
  - As ovulation takes place in between 12^{th} -16^{th} day of cycle and ovum has life span of 24 hours and sperm capacity to fertilise for 24 hrs. The fertile period will be from 9^{th} to 17^{th} day of cycle. So safe period is from 1^{st} to 8^{th}, then 18^{th} to 28^{th} day.
  - The 1^{st} unsafe day is obtained by subtracting 20 days from the length of the shortest cycle and last unsafe day by subtracting 10 days from the longest cycle.

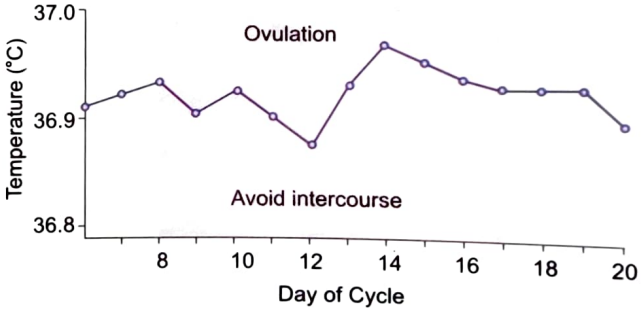


Fig. 16.1: Safe period

  b) Noting the basal body temperature chart (temperature rhythm) - Abstinence is practiced until the 3^{rd} day of the rise of temperature.

  c) Mucus rhythm (excessive mucoid vaginal discharge) - Abstinence on all the days plus 3 days thereafter from noticeable mucus production.

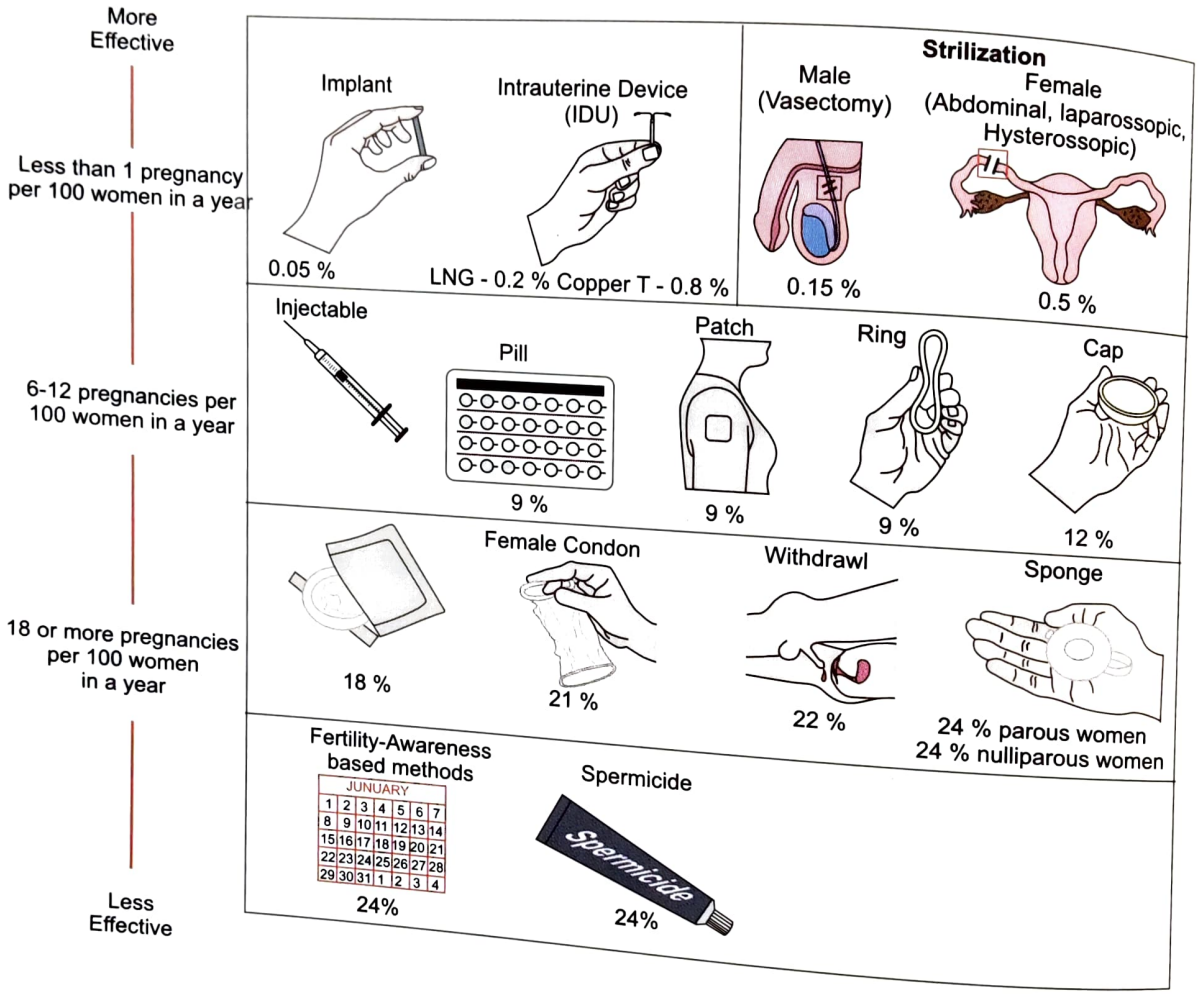


Fig. 16.2: Pearl index of various contraceptive methods

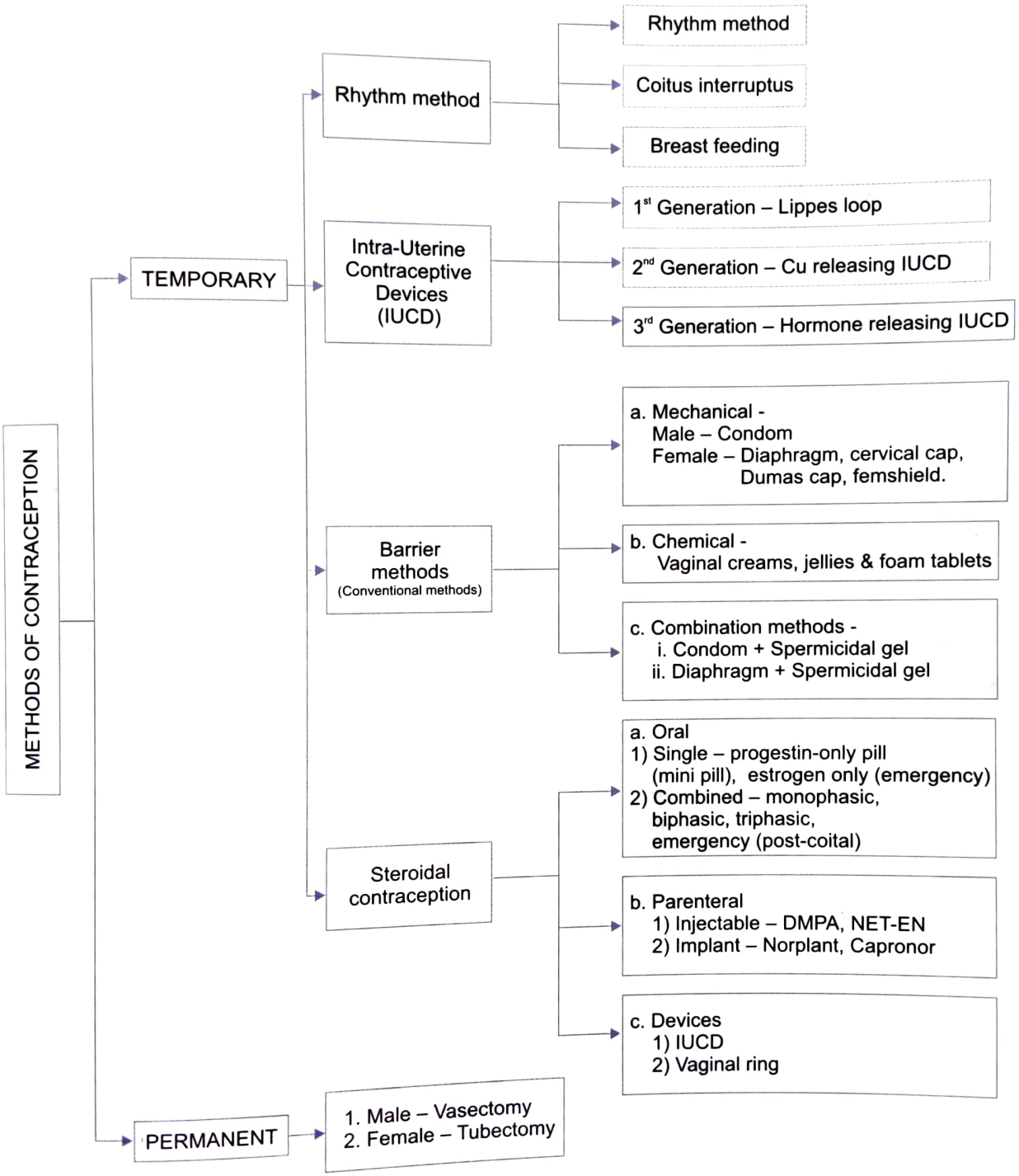


Fig. 16.3: Methods of contraception

- **Advantages**
  1. No side effects
  2. No expense.
- **Disadvantages**
  1. Not reliable
  2. Difficult to calculate
  3. Not applicable
     - In lactation amenorrhea
     - In irregular periods
  4. Complete abstinence needed in fertile period
  5. Higher rate of ectopic pregnancy
  6. Higher rate of congenital abnormalities, due to fusion of aging sperm and ova.
- **Coitus interruptus** or withdrawal of the penis from the vagina before ejaculation is one of the oldest methods of contraception. With this method, the majority of semen is deposited outside of the female reproductive tract with the intent of preventing fertilization.
- **Breast feeding (Lactational amenorrhea)**

  After delivery, the restoration of ovulation is delayed because of a nursing-induced hypothalamic suppression of ovulation. Continuation of nursing has long been a widespread method of contraception for many couples.

INTRA-UTERINE CONTRACEPTIVE DEVICES (IUCD)

- **1st Generation** – Lippes loop
- **2nd Generation** – Cu releasing IUCD
  a. This IUD causes a local, sterile inflammatory reaction in the uterus and the intrauterine environment becomes spermicidal.
  b. The copper intensifies the inflammation in the uterine cavity, producing a lining that is unfavourable for implantation.
- **3rd Generation –** Hormone releasing IUCD
  a. This IUD exerts its contraceptive effect locally on the endometrium and the cervix. Progestin alters the endometrium, rendering it unfavorable for implantation.
  b. In addition, both uterine and tubal motility are impaired, thereby impairing sperm-egg interaction. Thickening of the cervical mucus makes the passage of sperm difficult.
- **Patient selection**

  IUCD is preferred in woman who have
  a. A low risk of STD

b. Are multiparous

c. Are in a monogamous relationship

d. Want a long term reversible method of contraception

e. Unsatisfied with oral or barrier contraceptives.

➢ **Mechanism of action**

1. Action on uterus

   a) Acts as a foreign body and renders the migration of spermatozoa difficult.

   b) Provokes increased uterine contractility and increased tubal peristalsis - propulsion of ovum more rapidly to uterus before it is receptive.

   c) Leucocytic infiltration in endometrium - engulf the fertilized ovum.

2. Impair sperm ascent.

3. Cu-T devices causes enzymatic and metabolic changes in endometrium - gametotoxic and spermolytic effect.

4. Progesterone devices thickens the cervical mucosa and inhibits sperm ascent.

➢ **Advantages**

Cu releasing IUCD:

a. As many as 10 years of contraceptive efficacy from one IUD

b. Can be inserted at any time during the menstrual cycle

c. Resumption of fertility on removal.

Hormone releasing IUCD

a. As many as 5 years of contraceptive efficacy from one IUD

b. Useful for treatment of menorrhagia (heavy menstrual bleeding) and dysmenorrhea (painful menses)

c. Resumption of fertility on removal of the IUD.

➢ **Disadvantages**

1. Insertion - Although the copper-impregnated IUD may be inserted at any time during the menstrual cycle, the progestin-only IUD should be inserted within the first 7 days.

2. Uterine perforation.

3. Infection.

➢ **Efficacy –** Pregnancy rate among users of either type of IUD is less than 2 to 3 in 100 women-years of use.

➢ **Advice following insertion** – Patient is instructed to come for follow-up after the first cycle and every 6 months.

She is asked to feel the thread herself once in 3 months.

➢ **Contraindications:**

Absolute contraindications:

- Current pregnancy

- Undiagnosed abnormal vaginal bleeding
- Suspected gynecologic malignancy
- Acute cervical, uterine or salpingeal infection
- History of PID.

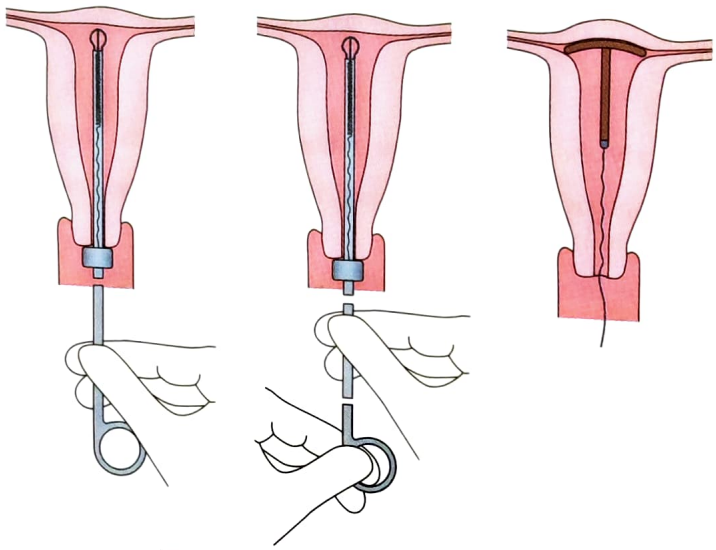


Fig. 16.4: Insertion IUD (Cu-T)

Relative contraindications:

- Nulliparity or desire for future childbearing
- Prior ectopic pregnancy
- History of STDs
- Multiple sexual partners
- Moderate or severe dysmenorrhea
- Congenital malformation of the uterus.

➢ **Translocated Cu- T:**

- Caused by perforation, expulsion or enlargement of uterus due to pregnancy.
- Diagnosed by non-visibility of the thread through the external os and confirmed by USG, hysteroscopy, sounding of the uterine cavity and if negative – straight X-ray after introducing a radio-opaque uterine sound into the uterine cavity.
- Management – If intrauterine, removed using Shirodkar's IUCD hook or by hysteroscopy. If extrauterine, then laparoscopy or laparotomy (rarely needed).

➢ **Levonorgestrel releasing device (LNG-IUS/Mirena/Levonova)**

- Contains a column of levonorgestrel (52-60 mg) within a rate-limiting membrane wrapped around the stem of the Nova-T frame.
- It releases a very low dose of hormone (20 mcg/day).
- Efficient for 5 years with a pregnancy rate of 0.7 per 100 woman years.
- Menstrual blood loss is markedly reduced and is recommended for the management of menorrhagia.

BARRIER METHODS

a. **Mechanical**

Condoms:

1. Mode of action – both female and male condoms act as physical barriers to semen.
2. Advantages:
   a. Protection from sexually transmitted diseases (STDs) and HIV
   b. Easy accessibility
   c. Few side effects
   d. Prevents sperm allergy
   e. No adverse effects on fetus if method fails.
3. Disadvantages:
   a. Must be used in each act of intercourse
   b. Unacceptability by some partners.
4. Types of condoms:
   a. Female condoms line the entire surface of the vagina and partially shield the perineum. They can be inserted upto 8 hours in advance but should be removed immediately after each act of intercourse. Female condoms should not be used in conjunction with male condoms.
   b. Male condoms cover the glans and the shaft of the penis and must be used from the beginning to the end of each act of intercourse to be effective.
5. Efficacy – Pregnancy rate is 10-14/100 women years of use.

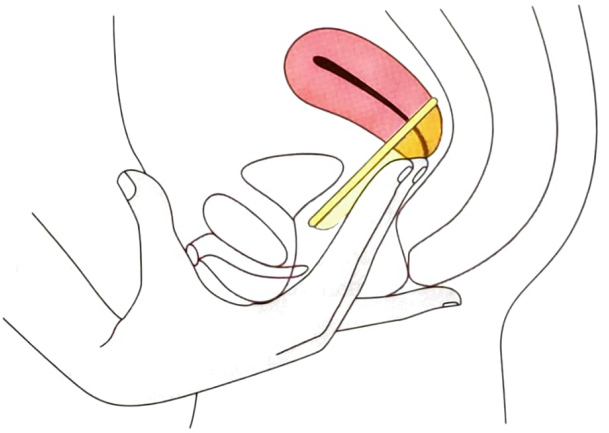


Fig. 16.5: Female condom

Diaphragms (Dutch cap)

These dome-shaped shaped contraceptives are 50 to 105 mm in diameter and are made of latex rubber. They rest between the posterior aspect of the symphysis pubis and the posterior fornix of the vagina, thus covering the anterior vaginal wall and the cervix.

1. Mode of action – Diaphragms act as physical barriers to sperm and are effective vehicles for holding spermicide over the cervix.
2. Advantages:
   a. Contraception for upto 6 hours after placement
   b. Some protection against cervical dysplasia
   c. Few side effects.
3. Disadvantages:
   a. Need to keep in place for at least 6 hours after intercourse to ensure that no motile sperms are left in the vagina
   b. Requirement for use with a spermicidal agent

c. Need to replace spermicide with each act of intercourse
d. Twice the incidence of urinary tract infections
e. Needs to be fit by a clinician.

4. Efficacy – Pregnancy rate is reported to be 9-30/100 woman years of use.

Cervical caps.

These contraceptives are as effective as diaphragms but are more difficult to fit.

1. Mode of action – The cervical cap has the same mode of action as the diaphragm.
2. Advantages:
   a. Ability to leave in place upto 48 hours (compared to 6 hours with the diaphragm), regardless of number of acts of intercourse
   b. Effectiveness without the addition of a spermicidal agent.
3. Disadvantages:
   a. Need to keep in place for at least 6 hours after intercourse (same as the diaphragm)
   b. Few physicians are trained in the placement and use of the cervical cap.
4. Efficacy – Pregnancy rate is reported to be about 9-30/100 woman years of use.

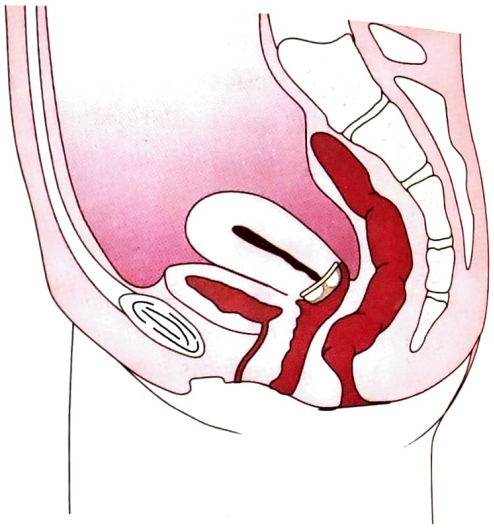


Fig. 16.6: Cervical cap

b. Chemical

Creams, jellies, aerosol foams, non-foaming and foaming suppositories and vaginal films are commonly used with other forms of contraception, such as diaphragms, sponges and condoms.

1. Mode of action – Spermicides serve as a chemical barrier to sperm. The active agents in spermicides disrupt the outer lipoprotein surface layer of spermatozoa, killing the sperm, decreasing their motility or inactivating the enzymes needed to penetrate the ova.
2. Advantages:
   a. Increased efficacy of vaginal sponges, diaphragms and cervical caps
   b. Easy accessibility
   c. No need for medical consultation
   d. Few side effects.
3. Disadvantages:
   a. Need for insertion with each act of intercourse
   b. Limited time of effectiveness
   c. Possible increase in the risk of HIV transmission caused by disruption of vaginal epithelium.
4. Efficacy – Pregnancy rate is as much as 30 in 100 women years of use. Efficacy depends on the couple's motivation to use spermicides correctly with every act of intercourse.

STEROIDAL CONTRACEPTION

a. Oral

1. Combined OCP's

a. Combined oral contraceptives – made up of Ethinyl estradiol + Progesterone.

b. Preparation:

Mala N - Progesterone (Norgestrol) 0.30 mg + Ethinyl oestradiol 30 g

Mala D - Norgesterol mg + Ethinyl oestradiol 30 g.

c. Time of consumption:

- 3 weeks on and 1 week off. Except Mala -N.
- Pill should be started on 1st day of menstrual cycle, 1 tablet/day for 21 days - stop for 1 week, then again start the next kit.
- In Mala-N – 28 tablets kit – 7 tablets are iron tablets.
- Should be taken from 21st to 28th day of cycle.

d. Mechanism of action:

1. Suppression of ovulation – Oral contraceptives place the body in a "pseudopregnancy" state by interfering with the pulsatile release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the anterior pituitary. This pseudopregnancy state suppresses ovulation and prevents pregnancy from occuring.
2. Inhibits ovulation.
3. Atrophic changes in endometrium – prevents nidation of fertilized ovum.
4. Progesterone makes cervical mucosa thick and prevents sperm ascent.

2. Monophasic pill

a. Progestin only pill – "Minipill"

b. Mode of action:

1. Diminishing and thickening cervical mucus, thereby preventing sperm penetration
2. Inhibiting ovulation by suppressing the midcycle peaks LH and FSH
3. Producing a thin, atrophic endometrium, precluding implantation
4. Reducing the ciliary action of the fallopian tube, preventing sperm and egg transport
5. Diminishing the function of the corpus luteum.

c. Advantages:

1. No alteration of milk production and nearly 100% effectiveness in breastfeeding women
2. Tolerance in women who are unable to take estrogen
3. Independent of sexual intercourse.

d. Disadvantages:

1. Irregular vaginal bleeding
2. No protection against STDs
3. Need for daily administration.

Efficacy– Pregnancy rate is 2-3 in 100 woman years of use.

3. Triphasic pill

a. The triphasic pill is a type of temporary method of steroidal contraception. It is made of ethinyl estradiol and levonorgesterol.

1st Phase → first 6 days → 30 g EE^2 + 50 g LNG

2nd Phase → next 6 days → 40 g EE^2 + 75 g LNG

3rd Phase → next 10 days → 30 g EE^2 + 125 g LNG.

b. Time of consumption – Tablet should be started from 1st day of menstrual cycle upto 21st day and break for 1 week.

c. Advantages:

1) It prevents breakthrough bleeding.
2) Minimal effect on lipid metabolism.
3) No adverse effect on carbohydrate metabolism – can be prescribed to diabetic patients.
4) Less side effects due to balanced estrogen-progesterone combination

d. Side effects:

a) All the side-effects of combined OCPs except few mentioned above in advantages of triphasic pills.

➢ Merits (of contraceptive pills)

1. It is highly effective
2. Failure rate is only 0.1/HWY
3. Good cycle control
4. Less side effects – well tolerated
5. Triphasic pills – lipid friendly
6. Progesterone only pill – decreased risk of ovarian and uterine cancer
7. Reversibility rate is high
8. Prevents anemia
9. Decreased endometriosis
10. Prevents against rheumatoid arthritis
11. Prevents dysmenorrhea and pre-menstrual tension
12. Decreased risk of PID
13. Free supply through government.

➢ Demerits

1. Compliance
2. Requires initial check up and periodic supervision
3. Side-effect of oral pills – as mentioned below.

Side effects of oral contraceptives are:

1. Due to progesterone:
   a) Acne
   b) Weight gain
   c) Amenorrhea/ Hypomenorrhea
   d) Libido
   e) Hyperglycemia
   f) Cholestatic jaundice

g) Increases the incidence of UTI
h) Leg cramps.

2. Due to estrogen:
   a) Nausea, vomiting and headache
   b) Chloasma
   c) Menstrual irregularity
   d) Leucorrhea
   e) Depression, sleep disturbances, psychiatric manifestations
   f) Hypertension
   g) Thromboembolic phenomenon
   h) Carcinoma endometrium, breast carcinoma, hepatocellular adenoma
   i) In case of fibroid uterus – it causes red degeneration of fibroid
   j) Reduces liver function capacity.
3. Combined effect:
   a) Mastalgia
   b) Monilial infection
   c) Disturbs hypothalamo-pituitary axis
   d) Hypoplasia of cervix
   e) Breast milk – reduction of protein and fat.

b. Parenteral

A. Injectable

a. Mode of action
   1. Continuous release of progesterone suppresses the LH surge and ovulation.
   2. Thickening of cervical mucus prevents sperm penetration.

   (a) Depo medroxy progesterone acetate (DMPA or depo-provera) in a dose of 150 mg every 3 months (micro crystalline aqueous suspension) by deep intramuscular injection been used with considerable efficacy.

   * Mode of action of DMPA:
     - Ovulation suppression
     - Thickening of cervical mucus preventing sperm penetration
   * Timing of administration:
     - Day 1-2 of the cycle
     - Postpartum 6 weeks after delivery
     - Post MTP/ miscarriage – can be given within the first 7 days.
   * Advantages:
     - Effective, convenient and reversible
     - Lower incidence of PID and ectopic pregnancy
     - Amenorrhea often occurs with less anemia, dysmenorrhea and premenstrual tension.
   * Side effects:
     - Weight gain
     - Delay in resumption of fertility of upto 1 year.

(b) Norethisterone enanthate (NET-EN) 200 mg every 8 weeks (in castor oil solution)

After withdrawal, the contraceptive effect of DMPA lasts longer than NET-EN.

Pregnancy rate is 1 per 100 woman years for DMPA and 0.6 per 100 woman years for NET-EN

B. Implant

- Norplant

Norplant is a temporary reversible method of steroidal contraception. It is made up of levonorgestrol (progesterone).

* Types:

a) Norplant I contains 6 capsules – each 36 mg of levonorgestrol. It releases 50 g/day for 5 years

b) Norplant II contains 2 rods – each 70 mg - releases 50 g/day for 3 -5 years (Capsule is made up of siloxane material).

* Insertion:

a) Inserted 1st day of

1. Menstrual cycle
2. Immediately after abortion
3. 3 weeks after post partum.

b) Inserted intradermally into inner aspect of non-dominant arm above elbow fold.

* Mechanism of action:

a) Inhibits ovulation

b) Thickens the cervical mucosa

c) Biochemical and enzymatic changes in endometrium.

* Removal – Removed within 5 years.

* Side Effects:

a) Irregular menstrual cycle, amenorrhea

b) Other side effects of progesterone like acne, chloasma, weight gain, libido

C. Devices

Vaginal ring —

a. This hormonal contraceptive contains ethinyl estradiol and etonogestrel (3-keto- desogestrel).

b. Use – The vaginal ring is worn continuously for 3 weeks and then discarded; a new ring is inserted into the vagina 1 week later. If the ring is left in place for more than 4 weeks, its contraceptive efficacy may be reduced.

c. Mode of action and efficacy are similar to combined OCPs.

d. Advantages

a. Contraceptive effect of 3 weeks

b. Beginning of contraceptive effect within the first day of use

c. Independent of sexual intercourse

d. Rapid resumption of fertility on discontinuation.

e. Disadvantages include lack of protection from STDs.

EMERGENCY (POST COITAL CONTRACEPTION)

- **Indications**
  1. Contraceptive failure. Eg. ruptured or slipped condom, misplaced diaphragm or a forgotten pill.
  2. Failure to use a contraceptive (unprotected intercourse).
  3. Incorrect or inconsistent use of a regular contraceptive.
  4. Accidental intercourse due to rape, assault or sexual assault.
- **Methods**
  1. Oestrogen alone – Ethinyl estradiol 5 mg/ day or conjugated oestrogen, 30 mg per day. Treatment is continued for 5 days, beginning within 72 hrs after an isolated act of coitus.
  2. Combined pill – Ethinyl estradiol 50 g + D-norgestrel 500 g (Ovral) given in doses of two tablets 12 hrs apart was initially tested in Canada by Yuzpe in 1977 and is known after him as the Yuzpe regime. Effectiveness is 75%, if taken within 24 hrs.
  3. Progestin only pills (POP) – Levonorgestrel is the only progestin studied for emergency contraception, one tablet of 0.75 mg LNG is taken within 72 hrs of coitus and repeated after 12 hours. Available as Pill 72 (Cipla) in India. This regime is highly effective (95%) in preventing unwanted pregnancies. It is well tolerated with a lower incidence of side effects like nausea and vomiting.
  4. Antiprogestogens – These are anti-implantation agents and can be given as late as 24^{th} – 27^{th} day of their cycle. RU 486 (mifepristone) has been found effective in a dose of 600 mg, when used as an emergency contraceptive. It acts by blocking the action of progesterone on the endometrium, causing sloughing and shedding of the decidua.
  5. Anti-oestrogens
     a. Danazol – has a direct luteolytic effect with a 400 mg dose repeated after 12 hrs for two to three doses.
     b. Centchroman (Saheli) is used as two 50 mg tablets given 12 hrs apart within 2 hrs of the act of coitus. It acts on the zygote, affecting ovum transport and blastocyst development.

FEMALE STERILIZATION - TUBECTOMY

- **Definition –** Tubectomy is an operation where resection of a segment of both the fallopian tubes is done to achieve permanent sterilization.
- **Time of Operation**
  1. Puerperal/postpartum – If the patient is otherwise healthy, the operation can be done 24-48 hours following delivery. Its chief advantage is technical simplicity. Hospital stay and rest at home following delivery are enough to help the patient to recover simultaneously from the 2 events.
  2. Interval – The operation is done beyond 6 weeks following delivery or abortion. The ideal time is following the menstrual period in the proliferative phase.
  3. Concurrent with MTP – Sterilization is done along with MTP. This is mostly done in the urban centers.
- **Methods**
  1. Laparotomy – Incision >5 cm, transperitoneal approach under GA/SA. In conjunction with other surgery (Caesarean section, salpingectomy, ovarian cystectomy, etc).
  2. Mini-laparotomy – Through a small suprapubic incision:

a. Pomeroy's method – A loop of fallopian tube is tied with a single absorbable suture at the base and loop excised. Failure rate is 0.4% due to recanalization.

b. Modified Irving method – Middle portion of the tube is tied. The intervening portion is excised, the proximal end buried in the uterus and distal end in the broad ligament. An irreversible method.

c. Madlener method – A loop of tube is crushed and ligated with non-absorbable suture. No tube is excised.

d. Aldridge method – A hole is made in the anterior leaf of broad ligament and the fimbrial end is buried into this.

e. Uchida method – Proximal end is buried in the broad ligament and distal end is ligated.

f. Modified Pomeroy's (Parkland) – The midportion of the tube is excised after ligating the segment with two separate absorbable sutures.

g. Kroner's fimbriectomy – Fimbrial end of the tube is double ligated and excised.

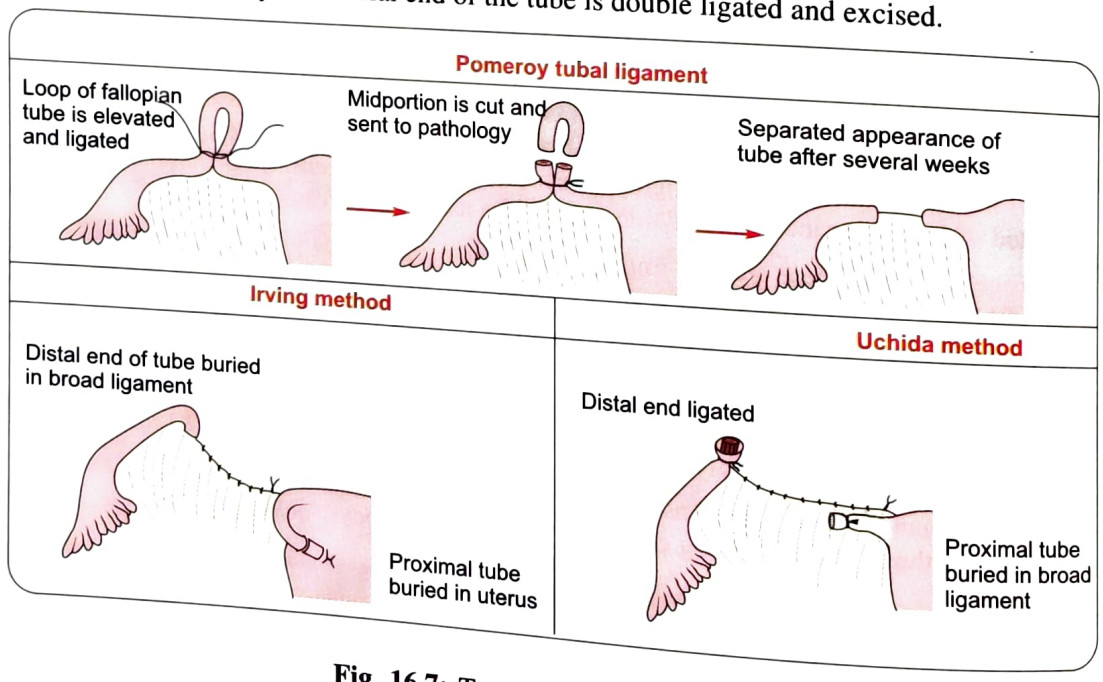

Pomeroy tubal ligament
Loop of fallopian tube is elevated and ligated
Midportion is cut and sent to pathology
Separated appearance of tube after several weeks
Irving method
Distal end of tube buried in broad ligament
Proximal tube buried in uterus
Uchida method
Distal end ligated
Proximal tube buried in broad ligament

Fig. 16.7: Types of Tubectomy

3. Vaginal route – vaginal approach by colpotomy is used for tubal ligation.
4. Laparoscopy:
   - Fibre-optic light source with laparoscopy allows access to the fallopian tubes with one sub-umbilical puncture.
   - Pneumoperitoneum is first created using carbon dioxide and fallopian tubes are identified.
   - Mechanical occlusion devices such as the spring-loaded clip (Hulka clip) or a Silastic rubber band (Falope ring) are used for occlusion of the tubes.
   - Advantages – Quicker recovery, can be performed on outpatient basis, effective right away and reversal more likely to succeed due to minimal damage to oviductal tissue.
   - Immediate complications of laparoscopic tubal ligation include the following:
     1. Incorrect anatomical site of sterilization
     2. Bowel, bladder, or blood vessel injury or perforation

3. Conversion to laparotomy
4. Electrocautery complications
5. Transection of the fallopian tube
6. Uterine perforation or cervical laceration

5. Hysteroscopy – Utero-tubal junction is occluded through a hyteroscope. High failure rates.

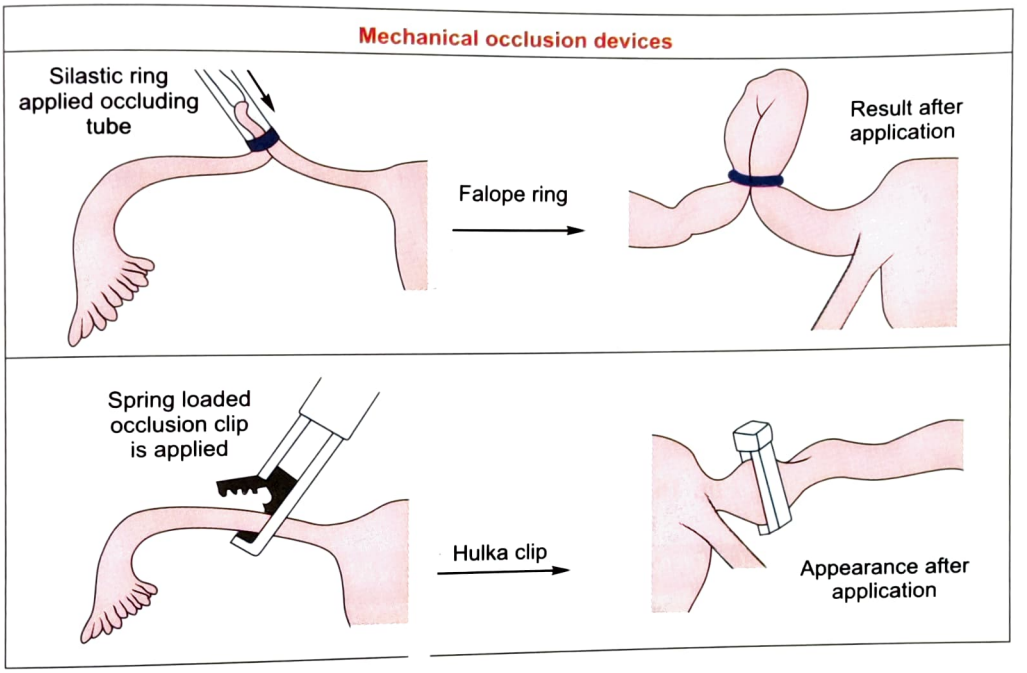


Fig. 16.8: Types of Laparoscopic tubal occlusion

MALE STERILIZATION - VASECTOMY

- **Definition:** Vasectomy is a procedure of occlusion or division of the vas deferens to prevent the passage of sperms.
- **Procedure:**

  The traditional incision approach of vasectomy involves numbing of the scrotum with local anesthetic after which a scalpel is used to make two small incisions on each side of the scrotum at a location that allows the surgeon to bring each vas deferens to the surface for excision. The vasa deferentia are cut (sometimes a piece removed), separated and then at least one side is sealed by ligating (suturing), cauterizing or clamping.
- **Precaution:** Sterility is not immediate since sperms are stored in the tract for upto 3 months. Patient is advised to use other methods of contraception during that period.
- **Complications:** Local wound infection, scrotal hematoma, sperm granuloma, spontaneous reanastamosis, anti-sperm antibodies, post-vasectomy syndrome (chronic intrascrotal pain and discomfort), increased risk of prostatic cancer.
- **No-scalpel vasectomy:** A specially designed instrument is used for isolating and delivering the vas through a small puncture in the skin.

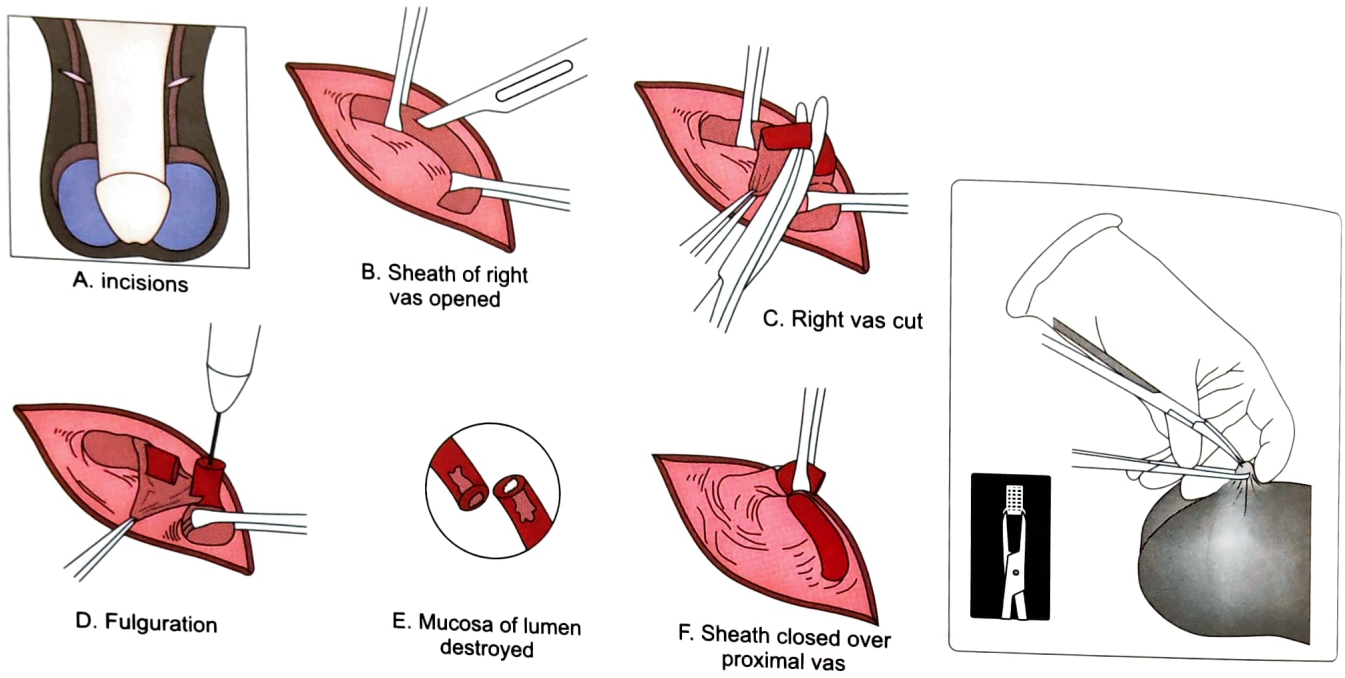

A. incisions
B. Sheath of right vas opened
C. Right vas cut
D. Fulguration
E. Mucosa of lumen destroyed
F. Sheath closed over proximal vas

Fig. 16.9: Procedure of vasectomy

MEDICAL TERMINATION OF PREGNANCY (MTP)

➢ **Indications**

1. Medical – where continuation of the pregnancy might endanger the mother's life or cause grave injury to her physical or mental health
2. Eugenic – where there is substantial risk of the child being born with serious handicaps due to physical or mental abnormalities
3. Humanitarian – where pregnancy is the result of rape
4. Socioeconomic – where actual or reasonably foreseeable environments (whether social or economic) could lead to risk of injury to mother's health
5. Failure of contraceptive devices – The anguish caused by an unwanted pregnancy resulting from a failure of any contraceptive device or method can be presumed to constitute a grave mental injury to the health of the mother. This condition is a unique feature of the Indian law and virtually allows abortion on request, in view of the difficulty of proving that a pregnancy was not caused by failure of contraception.

➢ **Methods of Termination of Pregnancy**

1. First Trimester (up to 12 weeks)
   a. Surgical
      1) Menstrual regulation
      2) Suction evacuation and/or curettage
      3) Dilatation and evacuation rapid method, slow method

b. Medical
   1) Mifepristone
   2) Mifepristone and misoprostol
   3) Methotrexate and misoprostol
   4) Tamoxifen and misoprostol.

2. Second Trimester (13-20 weeks)
   a. Surgical
      1. Amniotomy
      2. Intrauterine instillation of hyperosmotic solutions
         - Intra-amniotic – hypertonic urea, saline
         - Extra-amniotic – ethacrydine lactate, prostaglandins
   b. Medical
      1) Prostaglandins – misoprostol, carboprost, dinoprostone
      2) Oxytocin infusion – high dose used along with either of the above methods

➢ **Complications of MTP**

1. Gynecological Complications:
   a. Menstrual disturbances
   b. Chronic pelvic inflammation
   c. Infertility due to cornual block
   d. Scar endometriosis
   e. Uterine synechiae leading to secondary amenorrhea
2. Obstetric Complications:
   a. Recurrent midtrimester abortion due to cervical incompetence
   b. Ectopic pregnancy
   c. Preterm labor
   d. Dysmaturity
   e. Increased perinatal loss
   f. Rupture uterus
   g. Rh isoimmunization in Rh-negative women, if not prophylactically protected
   h. Failed abortion and continued pregnancy.

PREVIOUS EXAMINATION QUESTIONS

Long Essays

1. Mention various methods of contraception. Describe the advantages and disadvantages of oral contraceptive.
2. Describe different types of hormonal contraceptions. Its advantages and disadvantages.
3. A 24-year old primigravida approaches you for contraceptive advice. Discuss the merits and demerits of options you consider.
4. What is female sterilization? Describe different methods of doing it.

Short Essays

1. Describe natural family planning methods.
2. Contraceptive and non-contraceptive uses of deluxe condom.
3. Indications, contraindications and complications of IUCD.
4. Management of missing IUCD.
5. Name the different types of oral contraceptives. Mention modes of action and side effects.
6. Mechanism of action of oral contraceptives.
7. Mechanism of action and side effects of oral contraceptive pills.
8. Merits and demerits of contraceptive pill.
9. Enumerate contraindications for administration of oral contraceptive pills.
10. Merits and demerits of depot preparations for contraception.
11. Methods of emergency contraception.
12. Minilaparotomy for tubal ligation.
13. Advantages and complications of laparoscopic sterilization.
14. Contraindications for IUCD use
15. Barrier contraceptives.
16. Complications of IUCD.
17. Copper T.
18. Second generation IUCD.
19. Oral contraceptives.
20. Oral pill.
21. Combined contraceptives.
22. Merits and demerits of contraceptive pills.
23. Contraindications of oral contraceptives.
24. Side-effects of contraceptives.
25. Conventional contraceptives.
26. Oral contraceptives – side effects.
27. Mechanism of action and side effects of oral contraceptive pills.
28. Triphasic pills.
29. Progesterone only pill.
30. Progestogens and their application in gynaecology.
31. Non-steroidal oral contraception.
32. Norplant.
33. Methods of emergency contraception.
34. Interval sterilization.

Short Essays	35. Puerperal sterilization. 36. Tubal ligation. 37. Methods of termination of second trimester abortion. 38. Methods of MTP for second trimester. 39. Prostaglandins for abortion. 40. Laparoscopic tubectomy. 41. Enumerate injectable hormonal contraceptives, their advantages and disadvantages. 42. Enumerate the complications and contraindications of intra uterine contraceptive device. 43. Minilap tubectomy. 44. Intrauterine contraceptive devices. 45. How would you select a patient for LNG IUS? What are the uses of LNG IUS (intra uterine device)? 46. Hormonal contraception – its side effects. 47. Emergency contraception – advantages and disadvantages. 48. RU 486. 49. Diagnosis and management of missing IUCD. 50. Name the different types of oral contraceptives. Mention modes of action and side effects. 51. Merits and demerits of contraceptive pill. 52. Enumerate contraindications for administration of oral contraceptive pills. 53. Contraindications and beneficial effects of oral contraceptive pills. 54. Hormonal contraception – its side effects. 55. Vasectomy and its complications. 56. Tubal laparoscopic ligation and its complications. 57. Advantages and complications of laparoscopic sterilization. 58. Contraceptive advise to a para 1, living 1 who has delivered 6 weeks ago. 59. What are the complications of second trimester termination of pregnancy? What is manual vacuum aspiration? 60. Describe postpartum sterilization timing, technique and complications.
Short Answers	1. Pearl index–definitions and applications. 2. Define "safe period" for family planning use. 3. Safe period – its significance. 4. Interpretation of BBT chart. 5. Symptothermic method of contraception. 6. Management of burst condom in the unsafe period. 7. Barrier contraception. 8. Non-contraceptive benefits of condom. 9. Copper-T. 10. Hormone releasing IUCD. 11. Advice following IUCD insertion. 12. Complications of intrauterine contraceptive devices.

Short Answers

13. Methods to diagnose missing intrauterine device.
14. How will you manage missing IUCD?
15. List the beneficial effects of oral contraceptives.
16. Long term benefits of oral contraceptive therapy.
17. Non-contraceptive advantages of hormonal oral contraceptive pills.
18. Contraindications of oral contraceptive pills.
19. Progestogen only pill (mini pill).
20. Injectable contraceptive.
21. Emergency contraception.
22. Post-coital contraception methods.
23. Non-steroidal oral contraceptive.
24. Advantages and disadvantages of vasectomy.
25. No scalpel vasectomy.
26. Interval sterilization.
27. Minilap.
28. What is a modified Pomeroy's method? Mention four other surgical methods of sterilization.
29. Contraindications of laparoscopic sterilization.
30. Preparation of a patient and steps of laparoscopic tubal ligation.
31. What are the complications of laparoscopic sterilization?
32. Rules of MTP.
33. Enumerate methods of first trimester MTP.
34. Three methods of contraception in lactating women.
35. Mention methods used in second trimester termination of pregnancy.
36. Prostaglandins for abortion.
37. Mention six indications for D & C.
38. Enumerate the complications of first trimester MTP.

Disorders of Menstruation

CHAPTER 17

NORMAL MENSTRUATION

- Menarche – 10-16 years, average 12.5 years.
- Menorrhea (normal menstruation) – Regular cyclic uterine blood flow lasting 2-5 days with an interval of 21-35 days and blood loss of 30-60 ml.

DEFINITIONS OF MENSTRUAL IRREGULARITIES

- Oligomenorrhea – Uterine bleeding occurring at intervals >35 days
- Polymenorrhea – Uterine bleeding occurring at interval <21 days
- Amenorrhea – No bleeding for >6 months
- Menorrhagia – Prolonged(>5 days) or excessive (>80 ml) uterine bleeding at regular intervals
- Metrorrhagia – Uterine bleeding at irregular intervals superimposed on normal cyclical bleeding
- Menometrorrhagia – Prolonged or excessive uterine bleeding occurring at irregularly timed and frequent intervals
- Hypomenorrhea – Decreased uterine bleeding at regular intervals
- Hypermenorrhea – Increased uterine bleeding at regular intervals
- Intermenstrual bleeding (spotting) – Uterine bleeding of variable amounts occurring between regular menstrual periods.

PRE-MENSTRUAL TENSION

- **Definition –** Variable cluster of symptoms that appear to occur on a regular basis prior to each menstrual episode.
- **Etiology –** Unknown.
- **Symptoms**
  - Occur 7-10 days before menses and relieved by onset of menses
  - 7 days symptom free interval must be present in first half of cycle
  - Physical – edema, headache (cyclic), irritability, leg cramps, nausea and vomiting, visual changes (cyclic), weight gain (cyclic)
  - Psychological – irritability, anxiety, depression, sleep disturbance, appetite change, libido change, fatigue, suicidal ideation.

➢ **Treatment**

1. No proven beneficial treatment, only suggested treatment
2. Psychological support
3. Diet:
   - Decreased sodium, fluids, carbohydrates
   - Increased protein
   - Avoidance of caffeine and alcohol
4. Medications:
   - Vitamin - B_6 (pyridoxine)
   - Progesterone suppositories
   - Diuretics for severe fluid retention
   - NSAIDs for discomfort, pain
   - Evening primrose oil (linoleic acid)
   - Danazol (Danocrine)
   - SSRI antidepressants in selected cases
5. Regular exercise.

AMENORRHEA

➢ Primary amenorrhea is the absence of menses by 14 years of age in girls without appropriate development of secondary sexual characteristics or by 16 years of age regardless of secondary sex characteristic development.

➢ Secondary amenorrhea is the cessation of menses for a period of 6 months or a three-cycle interval in women who have been menstruating regularly.

PRIMARY AMENORRHEA

➢ **Types**

1. Hypergonadotropic – FSH more than 20 IU/L; LH more than 40 IU/L
2. Hypogonadotropic – FSH and LH less than 5 IU/L
3. Eugonadotropic – FSH and LH 5 to 20 IU/L.

➢ **Etiology**

A) Hypergonadotropic amenorrhea

1. Turner's syndrome
2. Premature ovarian failure
   a. This condition is defined as ovarian failure before 40 years of age
   b. Causes include:
      (1) Genetic (45X; 45X/ 46XX; 47XXY)
      (2) Autoimmune disease

(3) Infectious (e.g., mumps)

(4) Chemotherapy or radiation therapy

3. Gonadal dysgenesis (XY genotype, Swyer syndrome)
4. Resistant ovary syndrome
5. Enzyme deficiencies
   a. 17 α-hydroxylase deficiency
   b. Decreased aromatase enzymes.

B) Hypogonadotropic amenorrhea

1. Anorexia
2. Kallmann's syndrome
   - Deficient secretion of GnRH is associated anosmia or hyposmia. Kallmann syndrome involves the failure of olfactory axonal and GnRH neuronal migration from the olfactory placode in the nose.
   - Inheritance is either X-linked, autosomal dominant or autosomal recessive.
   - Symptoms and signs include primary amenorrhea, normal karyotype, infantile sexual development and the inability to perceive odours.
   - Possible coexisting features include bone and renal anomalies, cleft lip and palate, color blindness or hearing deficit.
3. Postpill amenorrhea

   Amenorrhea may occur 6 or more months after patients stop taking oral contraceptive pills or 12 months after they stop taking medroxyprogesterone
4. Medications or drugs

   Birth control pills may cause amenorrhea
5. Pituitary diseases
   a. Craniopharyngioma
   b. Adenomas.

C) Eugonadotropic amenorrhea

1. Disorders of androgen excess – polycystic ovary syndrome
2. Disorders of the outflow tract or uterus
   a. Mullerian agenesis (also known as Mayer-Rokitansky-Koster-Hauser syndrome)
   b. Androgen insensitivity (also known as testicular feminization)
   c. Asherman syndrome
   d. Infection – Tuberculosis and schistosomiasis may cause amenorrhea, usually secondary to intrauterine scarring and adhesions.

➢ **Investigations**

- Ultrasound abdomen and pelvis to assess the presence/absence of uterus/ovaries.
- Buccal smear – presence of Barr body (sex chromatin) indicates normal 46XX karyotype.
- Serum FSH, LH, prolactin, oestradiol – a low gonadotropin level indicates a hypothalamo-pituitary cause, a high level indicates an ovarian cause (low estrogen levels lead to absence of negative feedback inhibition).
- Chromosomal analysis (45X0 in Turner's syndrome).
- Plain X-ray skull reveals widening of the sella in pituitary tumors.
- Contrast enhanced CT head (CECT) may reveal a pituitary tumour or craniopharyngioma.

➢ **Clinical approach based on findings regarding breasts and uterus:**

Breasts present, uterus present – Differential diagnosis includes an imperforate hymen,vaginal septum, anorexia nervosa, excessive exercise, and possible pregnancy before first menses.

Breasts present, uterus absent – Differential diagnosis is M llerian agenesis (Rokitansky-Kuster-Hauser syndrome) and complete androgen insensitivity (testicular feminization). Testosterone levels and karyotype help make the diagnosis.

Breasts absent, uterus present – Differential diagnosis is gonadal dysgenesis (Turner's syndrome) and hypothalamic-pituitary failure. FSH level and karyotype help make the diagnosis.

➢ **Management**

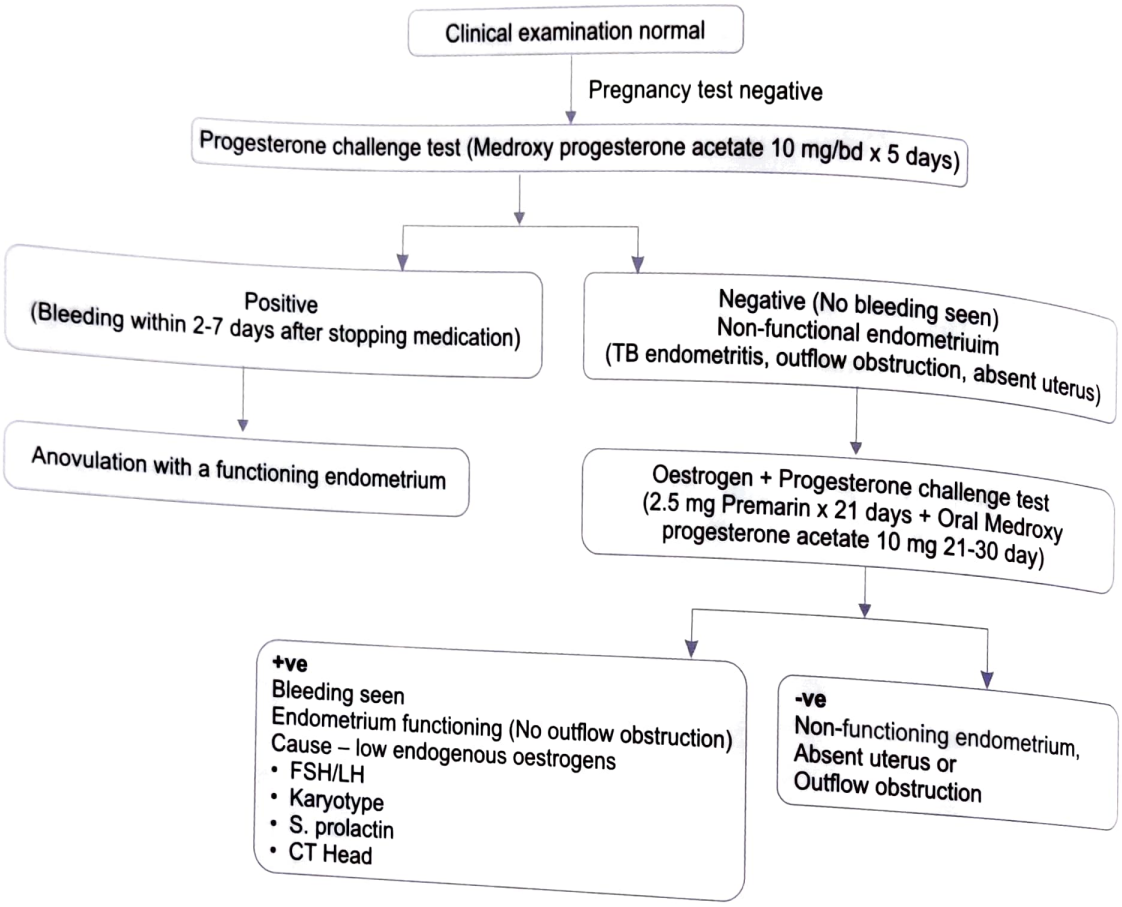
1. Correction of underlying cause
   - Surgical excision of craniopharyngioma and pituitary tumours.
   - XY karyotype – surgical removal of testicular tissue is indicated to prevent the development of gonadoblastoma.
   - Imperforate hymen – cruciate incisions are given in the hymenal membrane to relieve the collected menstrual blood.
   - Vaginal atresia/ septate vagina – vaginoplasty is indicated.
2. Correction of sexual infantilism – Exogenous estrogen + progesterone are given for development of breast and uterus.

SECONDARY AMENORRHEA

- **Definition** – Amenorrhea of 6 months or more in a woman with previous normal menstrual patterns in absence of pregnancy.
- **Causes**

1. Physiological:
   a. Pregnancy
   b. Lactation.
2. Pathological:
   a. Genital tract
      1) Acquired obstruction (gynatresia)
      2) Asherman syndrome.
   b. Ovaries
      1) Surgical extirpation
      2) Radiotherapy
      3) PCOD
      4) Autoimmune disease.
   c. Nutritional
      1) Anorexia nervosa
      2) Bulimia nervosa
      3) Obesity.
   d. Pituitary
      1) Insufficiency – Sheehan's syndrome
      2) Hyperprolactinemia
      3) Tumors – prolactinoma.
   e. Hypothalamus
      1) GnRH deficiency
      2) Pseudocyesis
      3) Vigorous exercise.
   f. Suprarenal
      1) Addison's disease
      2) Adrenogenital syndrome
      3) Suprarenal tumors.
   g. Thyroid
      1) Hypothyroidism
      2) Grave's disease.
   h. Others
      1) Drugs – phenothiazine, anti-depressants, OCP
      2) Diabetes
      3) TB
      4) Renal disease
      5) Severe anemia.

➢ **Evaluation of a patient with amenorrhea**


Clinical examination normal
Pregnancy test negative
Progesterone challenge test (Medroxy progesterone acetate 10 mg/bd x 5 days)
Positive
(Bleeding within 2-7 days after stopping medication)
Negative (No bleeding seen)
Non-functional endometriuim
(TB endometritis, outflow obstruction, absent uterus)
Anovulation with a functioning endometrium
Oestrogen + Progesterone challenge test
(2.5 mg Premarin x 21 days + Oral Medroxy progesterone acetate 10 mg 21-30 day)
+ve
Bleeding seen
Endometrium functioning (No outflow obstruction)
Cause – low endogenous oestrogens
• FSH/LH
• Karyotype
• S. prolactin
• CT Head
-ve
Non-functioning endometrium,
Absent uterus or
Outflow obstruction

➢ **Management:**

Management of secondary amenorrhea depends on the cause identified:

1. **Hypothalamo-pituitary causes** - Identification and elimination of the stressful factors, removal of intracranial tumours may result in return of menses. Patients who have had brain injury (due to surgery or RT) and have low gonadotropin levels need ovulation induction with gonadotropins or pulsatile GnRH therapy to restore fertility.
2. **Ovarian causes**
   - Premature ovarian failure need cyclical estrogens and progesterones to protect the female skeleton from demineralization.
   - Virilizing ovarian tumors need excision.
3. **Uterine causes**
   - Hysteroscopic division of intrauterine adhesions with HRT is indicated in Asherman's syndrome.
   - A patient with missed abortion needs dilatation and evacuation under antibiotic cover.
4. **Systemic diseases** need to be managed, menstrual cycles resume when the organ functions are restored.

DYSMENORRHEA

- **Definition** – Painful cramping pain accompanying menstruation.
- **Types**
  1. Primary dysmenorrhea – not associated with any identifiable pelvic pathology; due to biochemical derangement
  2. Secondary dysmenorrhea – associated with organic pelvic pathology (fibroids, adenomyosis, endometriosis, PID).
- **Varieties**
  1. Spasmodic dysmenorrhea:
     a. Most prevalent
     b. Manifests as cramping pains
     c. Most pronounced on first and second day of menstruation.
  2. Congestive dysmenorrhea
     a. Manifests as increasing pelvic discomfort and pelvic pain a few days before menses begin and relieved by menstruation
     b. Thereafter, patient rapidly experiences relief in her symptoms
     c. Commonly seen in PID or endometriosis.
  3. Membranous dysmenorrhea
     a. Endometrium is shed as a cast at the time of menstruation
     b. Passage of the cast is accompanied by painful uterine cramps
     c. It is a familial condition.
- **Clinical Features –** primary dysmenorrhea is widely prevalent
  1. Uterine cramping
  2. Nausea and vomiting
  3. Backache
  4. Giddiness, syncope and fainting
  5. Diarrhea.
- **Investigations**
  1. Pelvic ultrasound
  2. CT and MRI, if indicated
  3. Diagnostic hysterosalpingography
  4. Diagnostic sonosalpingography
  5. Diagnostic hysteroscopy
  6. Diagnostic laparoscopy.
- **Treatment**
  1. Counselling and psychotherapy
  2. Medical therapy
     a. Analgesics – paracetamol
     b. Anti-spasmodics – hyoscine

c. Prostaglandin synthetase inhibitors – mefenamic acid, indomethacin
d. Glyceryl trinitrate
e. Progestogen-containing IUCD – Mirena, Progestasert
f. OCP administered cyclically.

3. Surgical therapy – It is indicated in:
   a. Failure of medical measures to provide relief
   b. Secondary dysmenorrhea to treat underlying pelvic pathology.

CRYPTOMENORRHEA

- There is periodic shedding of the endometrium and bleeding but the menstrual blood fails to come out from the genital tract due to obstruction in the passage.
- **Causes**
  1. Congenital:
     a. Imperforate hymen
     b. Transverse vaginal septum
     c. Atresia of upper-third of vagina and cervix.
  2. Acquired:
     a. Stenosis of the cervix following amputation, deep cauterization and conization
     b. Secondary vaginal atresia following neglected and difficult vaginal delivery.
- **Clinical Features –** Patients with history of any of the etiological factors, complain of:
  1. Amenorrhea dated back from the events
  2. Periodic lower abdominal pain
  3. Pelvic examination reveals offending lesion in vagina or cervix
  4. Uterus is symmetrically enlarged.
- **Treatment**
  1. Cruciate incision of the hymen and drainage of blood
  2. Dilatation of the cervix in stenosis
  3. Reconstructive surgery in cases of secondary vaginal atresia.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. Describe investigations and management of secondary amenorrhea. 2. Enumerate causes of congestive dysmenorrhea. What are the treatment options for pelvic endometriosis? 3. Discuss in detail the etiopathogenesis and the management of primary amenorrhea.
Short Essays	1. Causes of primary amenorrhea. 2. Cryptomenorrhea. 3. Cryptomenorrhea and management. 4. Primary dysmenorrhea – definition, management. 5. Differences between spasmodic and congestive dysmenorrhea. 6. How will you investigate irregular bleeding in a woman in reproductive age and describe the management of one of them. 7. Primary dysmenorrhea – definition and management. 8. Causes of primary amenorrhea. 9. Sheehan's syndrome. 10. Discuss briefly the differential diagnosis of primary amenorrhea. 11. Ovarian causes of amenorrhea. 12. Causes of secondary amenorrhea. 13. Dysmenorrhea. 14. What are the types of dysmenorrhea? Describe each of them briefly. 15. Secondary dysmenorrhea.
Short Answers	1. Mention various menstrual abnormalities. Define two of them. 2. Name abnormal menstrual patterns and describe each of them. 3. Causes of primary amenorrhea. 4. Define secondary amenorrhea. Mention two important causes. 5. Physiological amenorrhea. 6. Ovarian causes of secondary amenorrhea. 7. Sheehan's syndrome salient features and treatment. 8. Premenstrual syndrome symptoms and treatment. 9. Oligomenorrhea. 10. Causes of intermenstrual bleeding. 11. Membranous dysmenorrhea. 12. Dysmenorrhea. 13. Congestive dysmenorrhea–definition and treatment. 14. Define congestive dysmenorrhea and mention two causes. 15. Define dysmenorrhea. How will you treat primary spasmodic dysmenorrhea?

Menorrhagia and DUB

CHAPTER 18

MENORRHAGIA

- **Definition –** Prolonged (> 5 days) or excessive (> 80 ml) uterine bleeding at regular intervals.
- **Causes**
  1. General Diseases
     a. Blood dyscrasias
        1) Severe anemia
        2) Leukemia
        3) Coagulopathy
        4) Thrombocytopenic purpura
     b. Thyroid dysfunction hypothyroidism and hyperthyroidism in early stages
     c. Generalized TB.
  2. Local Pelvic Causes
     a. Uterus
        1) Fibroids
        2) Fibroid polyp
        3) Adenomyosis
     b. Ovaries
        1) Chocolate cyst
        2) Ovarian feminizing tumors
        3) PCOD
        4) Endometriosis
     c. Infections
        1) Salpingo-oophoritis

2) PID
3) Genital TB

d. Immediate puerperal and post-abortal periods.

3. Contraceptives
   a. IUCD – 5-10% incidence
   b. Post-sterilization – 15% incidence.
4. Iatrogenic – estrogen and progesterone administration, especially mini pill.

- **Investigations**
  1. Complete physical examination
  2. Complete blood count
  3. Bleeding and clotting time
  4. Thyroid profile
  5. Pelvic ultrasound
  6. Diagnostic hysteroscopy
  7. Diagnostic laparoscopy.
- **Treatment**
  1. General measures
     a. Proper diet
     b. Adequate rest during menses
     c. Oral administration of hematinics, vitamins and protein supplements
     d. Maintenance of a menstrual calendar noting duration and extent of blood loss.
  2. Treat the underlying cause.

ABNORMAL UTERINE BLEEDING (AUB)

- **Definition** – "Abnormal uterine bleeding" includes any bleeding which is abnormal in its degree and timing. 'Dysfunctional uterine bleeding', or "DUB", is abnormal bleeding which has no obvious pathology.
- **Gynecological causes:**

a. Increased bleeding with menses
   - polyps
   - adenomyosis
   - leiomyomata
   - endometriosis
   - intrauterine device (IUD).

b. Bleeding following a missed period
   - ectopic pregnancy
   - abortion (missed, threatened, inevitable, incomplete, or complete)

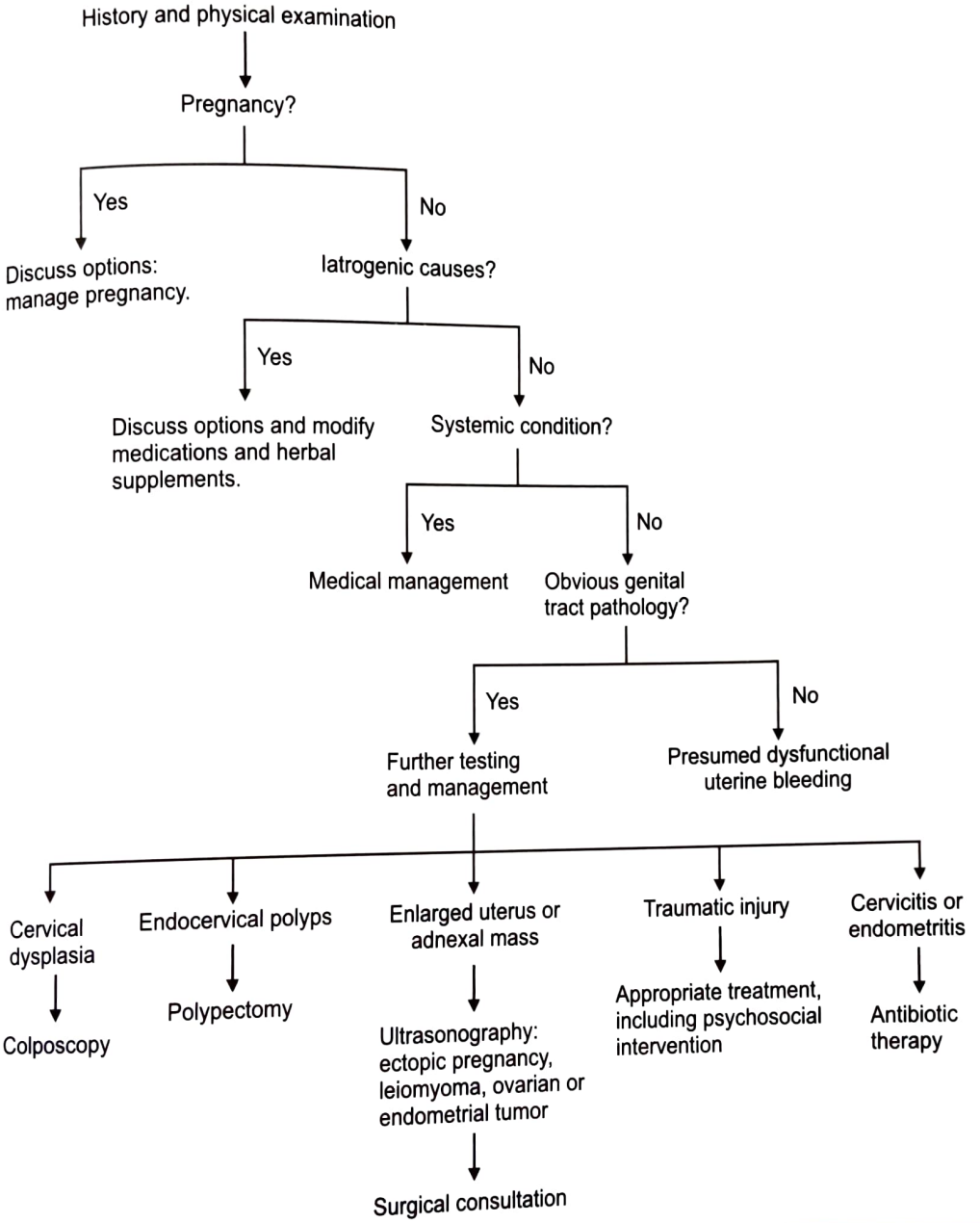
- implantation bleed
- trophoblastic disease.

c. Irregular bleeding
- dysfunctional uterine bleeding
- polycystic ovarian syndrome
- vulvovaginitis
- PID
- benign or malignant tumors of vulva, vagina, cervix, or uterus
- ovarian malignancy
- oral contraceptive use
- polyps.

2. Postmenopausal bleeding
- endometrial cancer until proven otherwise
- atrophic vaginitis (most common cause)
- ovarian malignancy
- benign or malignant tumors of vulva, vagina or cervix
- endometrial / endocervical polyps
- endometrial hyperplasia
- trauma
- polyps.

➢ **Non-Gynecological Causes**
- ➢ thyroid disease (hyperthyroid/ hypothyroid)
- ➢ chronic liver disease
- ➢ leukemia
- ➢ idiopathic thrombocytopenic purpura
- ➢ drugs: spironolactone, danazol, psychotropic agents
- ➢ metastatic cancer.

➢ **Management algorithm**



DYSFUNCTIONAL UTERINE BLEEDING (DUB)

- **Definition –** A state of abnormal uterine bleeding without any clinically detectable organic pelvic pathology.
- It is a diagnosis of exclusion, i.e., abnormal uterine bleeding which is not attributable to any of the following known causes:
  1. Pregnancy related complications – abortion, ectopic pregnancy

2. Tumors of uterus
   a. Benign – cervical polyp, endometrial polyp, fibroids
   b. Malignant – Ca. cervix, Ca. endometrium, Ca. fallopian tube
   c. Others – endometriosis, adenomyosis
3. Infections – PID, cervicitis, endometritis
4. Intrauterine foreign body – IUD
5. Systemic diseases:
   a. Hepatic – reduced coagulation factors, reduced metabolism of sex steroids
   b. Renal – reduced excretion of estrogens
6. Blood disorders:
   a. Platelet disorders – ITP, TTP
   b. Clotting factor disorders, leukemias, etc
7. Iatrogenic – exogenous hormones, anticoagulants
8. Endocrinal disorders – thyroid disorders, adrenal disorders.

➢ **Classification of DUB**

Anovulatory (80%)	Ovulatory (20%)
1. Threshold bleeding	1. Irregular ripening
2. Bleeding of puberty	2. Irregular shedding
3. Metropathia hemorrhagica	3. IUCD insertion
4. Premenopausal DUB	4. Following sterilization operation

➢ **Pathology:**

1. Anovulatory bleeding:
   - Unopposed estrogen and lack of progesterone
   - Excess endometrial growth → highly vascular and fragile
   - When estrogen withdrawal occurs, there is threshold bleeding with asynchronous endometrial shedding
   - Due to lack of progesterone → reduced ratio of $PGF_{2\alpha}$:PGE2 → heavy bleeding.
2. Ovulatory bleeding:
   a. Irregular endometrial ripening
      Inadequate corpus luteum function → inadequate progesterone secretion
      C/F – Premenstrual spotting/brownish discharge.
   b. Irregular endometrial shedding (Halban's disease)
      Persistent corpus luteal function → irregular desquamation of endometrium
      Persistent LH secretion → Continued progesterone secretion → No FSH → Inadequate estrogen secretion → Failure of endometrial regeneration.
   c. Metropathia hemorrhagica – excessive menses for long intervals associated with anovulatory cycles and cystic endometrial hyperplasia.

Type I – 8-10 weeks amenorrhea → continued bleeding for weeks
Type II – Previous cycles normal → continued bleeding at next cycle
Type III – Previous menorrhagia → continued bleeding
Important features
1. Parity not related to incidence
2. Age 40-45 years
3. Bleeding not severe but prolonged
4. Bleeding is always painless, since it is anovulatory
5. Thickening of endometrium + cystic ovary + myohyperplasia of myometrium
6. Bleeding is preceded by a short period of amenorrhea

➢ **Diagnosis**

1. History
   a. Bleeding – onset, duration, amount, character, cyclical features
   b. Antecedent cause – IUCD, recent delivery, abortion, drug therapy, sterilization operation
2. General examination
3. Abdominal examination
4. Pelvic examination
5. Complete blood count
6. Ultrasound – to exclude pelvic pathology
7. Diagnostic curettage – mainly to obtain material for histology
8. Hysterosalpingography – may reveal some intrauterine cause which was missed.

➢ **Management**

Medical management of DUB

A. General
   a. Rest
   b. Reassurance
   c. Anemia correction.

B. Hormonal
   - Estrogen – conjugate equine estrogen
   - Progesterone
     1. MPA 5^{th}-21^{st} day or 11^{th}-25^{th} day for 3-6 cycles [10 mg tid] – only to control bleeding
     2. Norethisterone
   - Combined – ethinylestradiol 0.25 mg + progesterone 10 mg
   - Androgens
     1. Danazol – 200-400 mg
     2. GnRH analogues – 3-6 mg (1 injection every month)

C. Non-hormonal
   - NSAIDs – mefenamic acid 500 mg tid
   - Antifibrinolytics – Tranexemic acid
   - Ethamsylate

- Clomiphene citrate 150 mg od 5th - 9th day.

Surgeries for DUB:

1. Hysterectomy
2. Endometrial ablation (TCRE – transcervical resection of endometrium)
3. B/L oopherectomy – surgery or radiotherapy
4. Uterine artery embolism.

Age	D&C + Hysteroscopy	Medical Line	Surgical (Hysterectomy / Ablation)
Adolescent age group	Only for refractory cases	1st choice always	Never
Reproductive age group	1st choice always	Always (as per HP report)	Rarely
Pre-menopausal age group	Mandatory	For a short time (palliative phase)	1st choice always

PREVIOUS EXAMINATION QUESTIONS

Long Essays

1. What are the causes of menorrhagia in a 35 year old woman and how will you manage a case of fibroid uterus in such a woman?
2. Define dysfunctional uterine bleeding and discuss the etiology. How do you manage a case of anovulatory dysfunctional bleeding in a forty year lady?
3. Define DUB. Briefly outline the management plan in a 40 year old woman with "DUB".
4. Discuss the management options in dysfunctional uterine bleeding.
5. Define DUB. Discuss the various options available to manage a 35 year old patient with DUB.
6. Write in brief the physiology of normal menstruation. Discuss the investigations in case of DUB.
7. How do you evaluate a case of DUB? Discuss management of metropathia hemorrhagica.
8. Management of pubertal DUB.
9. 30 year Para 2 living 2 (P2 L2) sterilized lady presents with menorrhagia responding to medical treatment. How would you evaluate her and what treatment would you offer – justify?
10. Define dysfunctional uterine bleeding. How do you evaluate and manage it in a 35 years old woman?
11. Define DUB. Discuss medical management in a 35 year old lady.
12. Discuss the diagnosis, investigations and management of a 45 year old multiparous woman coming with frequent and profuse bleeding during periods for the past 6 months.
13. Discuss the management of a P3L0 45 years old lady with abnormal uterine bleeding.

Short Essays	1. Causes of menorrhagia. 2. Define dysfunctional uterine bleeding and outline its medical management. 3. Puberty menorrhagia. 4. Differential diagnosis of abnormal bleeding in a pubertal girl. 5. Metropathia hemorrhagica. 6. Approach to the management of puberty menorrhagia. 7. Describe the management of adolescent dysfunctional uterine bleeding. 8. How does a patient present with anovulatory bleeding? How would you treat a woman at the age of 40 yrs with anovulatory DUB? 9. Describe the management of dysfunctional uterine bleeding in perimenopausal woman. 10. Non-hormonal management of dysfunctional uterine bleeding. 11. Medroxyprogesterone acetate.
Short Answers	1. Define menorrhagia. Mention two causes. 2. Enumerate the types of DUB. 3. Metropathia hemorrhagica characteristic features, management. 4. Endometrial pattern in DUB. 5. Ovulatory menorrhagia diagnosis and management.

Hormonal Therapy in Gynecology

CHAPTER 19

CLOMIPHENE CITRATE

- Clomiphene citrate is a non-steroidal compound related to diethylstilbestrol (DES). It is a mixture of 2 isomers, cis and trans. Cis fraction induces ovulation.
- **Mode of Action**
  1. Clomiphene citrate stimulates hypothalamic gonadotrophin (Gn) secretion by blocking the negative feedback of estrogen by the ovaries
  2. Gn stimulates pituitary FSH and LH secretion which induces ovulation.
- **Indications**
  1. Anovulatory infertility
  2. PCOD associated with infertility
  3. IVF – GIFT and ART
  4. To stimulate spermatogenesis.
- **Contraindications**
  1. Ovarian cyst – cyst can increase in size
  2. Chronic liver disease
  3. Scotoma.
- **Side Effects**
  1. Ovarian enlargement
  2. Hot flushes and sweating due to estrogen deficiency
  3. Nausea, vomiting
  4. Visual disturbances, blurring, scotoma
  5. Headache, dizziness
  6. Urticaria

7. Hair loss
8. Weight gain
9. Anti-estrogenic effect on cervical mucus
10. Ovarian hyperstimulation syndrome (OHSS).

HYPOTHALAMIC HORMONES

- **GnRH:** Gonadotrophin releasing hormone
  - Diagnostic – GnRH stimulation test is used to differentiate b/w amenorrhea of pituitary or hypothalamic origin.
  - Therapeutic – For activation of pituitary gonadal axis and induction of ovulation.
- **GnRH analogues:**
  a. They act both as agonists and antagonists.
  b. On chronic use initially they have effect of upregulation or flare effect and later they will show downregulation, which is due to reduced sensitivity of pituitary GnRH receptors.
  c. Pulsatile therapy overcomes downregulation and rather increases the receptor sensitivity.
  d. Various GnRH analogues used today are – buserelin, nafarelin, goserelin, leuprorelin and triptorelin.
- **Uses of GnRH analogues:**
  1. Controlled ovarian hyperstimulation in in-vitro fertilization
  2. Induction of ovulation with higher baseline LH as in PCOS
  3. Endometriosis
  4. Fibroid
  5. Precocious puberty
  6. Hirsutism
  7. DUB
  8. Premenstrual syndrome
  9. Advanced breast Ca
  10. Contraception in both male and female
  11. Endometrial resection/ablation.

  (Note: Long term use of GnRH analogues have menopause like symptoms due to hypoestrogen state.)
- **GnRH antagonists:** Ganirelix and Cetrorelix.
- **Gonadotrophins:**
  - The gonadotrophins used today are mainly obtained from urine obtained from postmenopausal women.
  - It is also obtained by recombinant technology today. Invariably both are costly.
  - hCG is obtained from urine of pregnant women.
  - Treatment is given under various protocols and it varies depending on whether the patient is a high or poor responder.
- **Anti-gonadotrophins:**
  - Danazol and gestrinone are the two main anti-gonadotrophins.
  - Basically they result in production of an hypoestrogenic state.

Indications:

- Endometriosis
- DUB
- Symptomatic fibroid
- Precocious puberty
- Premenstrual tension syndrome
- Benign fibrocystic disease of breast
- Prior to endometrial ablation

Its use has hypoestrogenic, androgenic and metabolic side effects.

GONADAL HORMONES

➢ **Estrogen:**

- They are available both in natural and synthetic forms.
- Synthetic forms – semisynthetic derivatives of oestradiol- ethinyl oestradiol and mestranol; and non-steroidal wholly synthetic preparations – diethylstilbestrol.
- Therapy:

  Replacement therapy – for hypo-ovarian function and menopausal symptoms.

  Pharmacotherapy – in oral contraception, vaginitis, intersex state, DUB, delayed puberty, cervical mucus hostility and as an adjuvant with clomiphene therapy.
- Contraindications of estrogen therapy:
  a. H/o venous thromboembolism
  b. Active liver disease
  c. Severe hypertension
  d. Organic heart disease
  e. Estrogen dependent tumors.
- Adverse effects are common and they are:
  a. Major – Increased incidence of endometrial Ca, thromboembolism, cerebral thrombosis and hemorrhage.
  b. Minor – Nausea, vomiting, breast tenderness, breakthrough bleeding and weight gain.

➢ **Anti-estrogen:**

They are clomiphene, letrozole and tamoxifen.

- Clomiphene – The commercially available form is a mixture of enclomiphene and zuclomiphene. In the hypothalamus it binds to estrogen receptors preventing the negative feedback effect of endogenous oestrogens to GnRH resulting in synthesis of more gonadotrophins.
- Letrozole – It is a specific aromatase inhibitor. It inhibits the enzyme aromatase to suppress synthesis of oestrogen. It enhances the secretion of pituitary FSH for the growth and development of follicles. It is used for the induction of ovulation in women with anovulatory infertility.
- Tamoxifen – Tamoxifen is also widely used in benign breast disease. Raloxifen is a more selective antioestrogen which is used in regression of endometriosis.

➢ **Progesterone:**

- Natural progesterone cannot be taken by oral route as it gets deactivated. The newer synthetic forms have overcome this deactivation and are successfully used orally.

- Synthetic forms:
  - Progesterone derivatives such as 17 α-hydroxyprogesterone caproate, medroxyprogesterone acetate, megestrol acetate and chlormadinone acetate.
  - Stereoisomers of progesterone such as dydrogesterone.
  - Testosterone derivatives such as ethisterone and dimethisterone.
  - 19- norsteroids such as norethisterone, norethisterone acetate, norethynodrel, allyloestrenol, ethynodiol diacetate and the 'newer' progestogens desogestrel, gestodene and norgestimate.
- Uses:

  Diagnostic:

  Progesterone challenge test in investigation of pathological amenorrhea.

  Therapeutic:

  a. Contraception
  b. DUB
  c. Endometriosis
  d. Primary dysmenorrhea
  e. Luteal phase defect
  f. Endometrial hyperplasia and endometrial Ca
  g. Premenstrual syndrome
  h. Postponement of menstruation – Norethisterone 5 mg twice daily (Primolut-N) is used
  i. Hormone replacement therapy.

➢ **Anti-progesterone:**

Mifepristone – It binds competitively to progesterone receptors and nullifies the effect of endogenous progesterone. RU 486 is newer form with long half life, active metabolites and higher affinity for the receptors.

Uses:

- Therapeutic abortion
- Postcoital contraception
- Induction of labor
- Uterine fibroids
- Ectopic pregnancy
- Cushing's syndrome.

➢ **Androgens:**

- It is used in cases of frigidity and premenstrual syndrome and also in male infertility.
- Some of the preparations available are phenyl propionate and methyl testosterone.

➢ **Anti-androgens:**

They are:

a. Cyproterone acetate – This compound blocks the hormone action at the receptor level. It is used in idiopathic hirsutism or hyperandrogenic states
b. Spirinolactone – It is an anti-androgenic diuretic. It inhibits androgen synthesis from ovary and adrenals.
c. Flutamide
d. Finasteride.

HUMAN CHORIONIC GONADOTROPIN

- hCG is a glycoprotein hormone which is produced during pregnancy by the embryo shortly after conception and later in large amounts, by the placenta (by the syncytiotrophoblast).
- The function of hCG during pregnancy is to maintain the corpus luteum and causing it to secrete progesterone.
- Uses:
  - Diagnosis of pregnancy using urine or blood sample.
  - To diagnose and monitor germ cell tumors and gestational trophoblastic diseases.
  - It is a component of the triple test, a screening test for certain fetal chromosomal abnormalities.
  - Stimulation of ovulation in females (often used in IVF).
  - Induce puberty in adolescent males.
  - Hypogonadism (defect of the reproductive system resulting in a lack of function of the gonads or ovaries)
  - As a performance enhancing drug in sports along with anabolic steroids.

PREVIOUS EXAMINATION QUESTIONS

Short Essays	1. Uses of estrogens in gynecology. 2. Clomiphene citrate. 3. hCG. 4. Artificial postponement of menses. 5. Ovarian hyperstimulation syndrome. 6. Indications for gonodotropin releasing hormone (GnRH) in gynecology.
Short Answers	1. Uses of progesterone in gynecology. 2. Danazol. 3. Clomiphene citrate–clinical uses. 4. RU 486. 5. Human chorionic gonadotropin (hCG).

Inflammation of the Uterus and Cervix

CHAPTER 20

CHRONIC CERVICITIS

- Chronic cervicitis is the commonest lesion found in clinical practice.
- Onset – It may follow an acute attack or usually be chronic from the beginning.
- Etiology – Infection usually occurs following childbirth or abortion by the pyogenic organisms – Staphylococcus, Streptococcus or E. coli.
- **Pathology**
  1. Mucosa and deeper tissues are congested, fibrosed and infiltrated with leucocytes and plasma cells
  2. Glands are hypertrophied with increased secretory activity
  3. Some gland mouths are closed by fibrosis or plugs of desquamated epithelial cells to cause retention cysts (Nabothian follicles).
- **Clinical Features**
  1. Symptoms:
     a. May be asymptomatic and accidentally discovered during examination
     b. Discharge – predominant symptom; excess mucoid discharge, at times mucopurulent usually dates from causative event (childbirth, abortion, etc.)
     c. Contact bleeding or associated infertility may be present.
  2. Signs:
     a. Cervix may be tender to touch or on movement
     b. Speculum examination reveals:
        1) Mucoid or mucopurulent discharge escaping out through cervical os
        2) May be enlargement, congestion or ectropion of the cervix
        3) Associated erosion may be present.
- **Treatment**
  1. Cervical scrape cytology to exclude malignancy is mandatory prior to any therapy

2. No place of antibiotic therapy except in gonococcal or chlamydial infection
3. Electrocauterization
4. Diathermy cauterization
5. Laser therapy
6. Cryosurgery
7. Trachelorrhaphy – reconstruction of associated cervical tear
8. Conization – in badly infected endocervix and external os
9. Hysterectomy – in unresponsive cases with patients approaching menopause.

CERVICAL EROSION (ECTOPY)

- **Definition** – Condition where the squamous epithelium of the ectocervix is replaced by columnar epithelium which is continuous with the endocervix.
- **Etiology**

1. Congenital – At birth, in about one-third of cases, the columnar epithelium of the endocervix extends beyond the external os. This condition persists only for a few days until the estrogen level derived from the mother falls. Thus, the congenital erosion heals spontaneously.
2. Acquired:
   a. Hormone – The squamo-columnar junction is not static. Its movement, either inwards or outwards is dependent on estrogen:
      1) When estrogen level is high, it moves out, so that the columnar epithelium extends onto the vaginal portion of the cervix, replacing the squamous epithelium
      2) This state is observed during pregnancy and amongst pill users
      3) Squamo-columnar junction returns back to normal after 3 months following delivery and a little earlier following withdrawal of the pill.
   b. Infection – The role of infection as a primary cause has been discarded. However, chronic cervicitis may be associated or the infection may supervene on an erosion because of the delicate columnar epithelium which is more vulnerable to trauma and infection.

- **Clinical Features**

1. Symptoms
   a. May be asymptomatic
   b. Vaginal discharge:
      1) Excessively mucoid – from overgrowth or overactive cervical crypts
      2) Mucopurulent, offensive and irritant - due to infection
      3) Blood-stained – due to premenstrual congestion
   c. Contact bleeding – especially during pregnancy and pill use either following coitus or defecation
   d. Associated cervicitis – backache, pelvic pain and at times infertility.

2. Signs – Speculum examination reveals:
   a. Bright red area surrounding and extending beyond external os in ectocervix
   b. Outer edge is clearly demarcated
   c. Lesion may be smooth or having small papillary folds
   d. Non-tender and non-friable
   e. On rubbing with a gauze piece, there may be multiple oozing spots
   f. Feel is soft and granular, which gives rise to grating sensation.

- **Treatment**

1. Cervical smear cytology to exclude malignancy is mandatory before treatment
2. In doubtful smear, colposcopy and/or cervical biopsy should be done
3. Asymptomatic cases do not require treatment
4. Detected during pregnancy and early puerperium – treatment should be withheld for at least 12 weeks postpartum
5. Pill users – pill should be stopped; barrier method is advised
6. Persistent erosion with troublesome discharge – surgical treatment:
   a. Thermal cauterization
   b. Cryosurgery
   c. Laser vapourization.

PYOMETRA

- It is the collection of pus in the uterine cavity.
- **Prerequisites** for its formation are:
  a. Occlusion of cervical canal.
  b. Enough sources for pus formation within uterus.
  c. Presence of low grade infection.
- **Causes**
  a. Obstetric – secondary to lochiometra.
  b. Gynecological – carcinoma in lower part of the body, endocervical Ca, senile endometritis, infected hematometra and TB endometritis.
- **Causative organisms:** *Coliforms, Streptococci, Staphylococci* and rarely tubercular.
- **Clinical Features**
  a. Intermittent, blood stained, purulent, offensive discharge PV.
  b. Pain per abdomen.
  c. Systemic manifestations absent.
- **Diagnosis** is by internal examination. US scan diagnosis mostly. It is confirmed by dilatation of cervix when

pus escapes. Malignancy must be ruled out. Diagnostic curettage must be withheld for 7-10 days to prevent perforation and hence spreading peritonitis.

- **Treatment** in simple non-malignant cases is by drainage with dilatation. In recurrent cases hysterectomy is indicated. Definite surgery for malignancy following drainage.

PREVIOUS EXAMINATION QUESTIONS	
Short Essays	1. Pyometra. 2. Management of erosion cervix. 3. Erosion of cervix.
Short Answers	1. Causes of pyometra and treatment. 2. Define cervical erosion and ectropion. 3. What is erosion of cervix and how do you manage? 4. What is erosion of cervix? What are its types? 5. Management of erosion of cervix. 6. Cold - knife conization.

Genital Prolapse

CHAPTER 21

SUPPORTS OF UTERUS

- **Primary Supports**
  1. Muscular (Active)
     a. Perineal body
     b. Pelvic diaphragm
     c. Urogenital diaphragm
  2. Fibromuscular (Mechanical)
     a. Uterine axis
     b. Uterosacral ligaments
     c. Pubocervical ligaments
     d. Transverse cervical ligaments of Mackenrodt
     e. Round ligaments of uterus.
- **Secondary Supports**
  1. Broad ligaments
  2. Uterovesical fold of peritoneum
  3. Rectovaginal fold of peritoneum.

GENITAL PROLAPSE

- **Definition**

  It is a condition of descent of uterus and vagina from their normal anatomical position either partially or completely.
- **Classification**
  1. Vaginal Prolapse
     a. Anterior vaginal wall
        i. Upper 2/3rd – Cystocele

ii. Lower 1/3rd – Urethrocele

b. Posterior vaginal wall

i. Upper 1/3rd – Enterocele

ii. Middle 1/3rd – Rectocele

iii. Lower 1/3rd – Lax perineum

2. Uterine prolapse

a. 1 – Descent of cervix into the vagina

b. 2 – Descent of cervix upto the introitus

c. 3 – Descent of cervix outside the introitus

d. Procidentia – entire uterus outside the introitus.

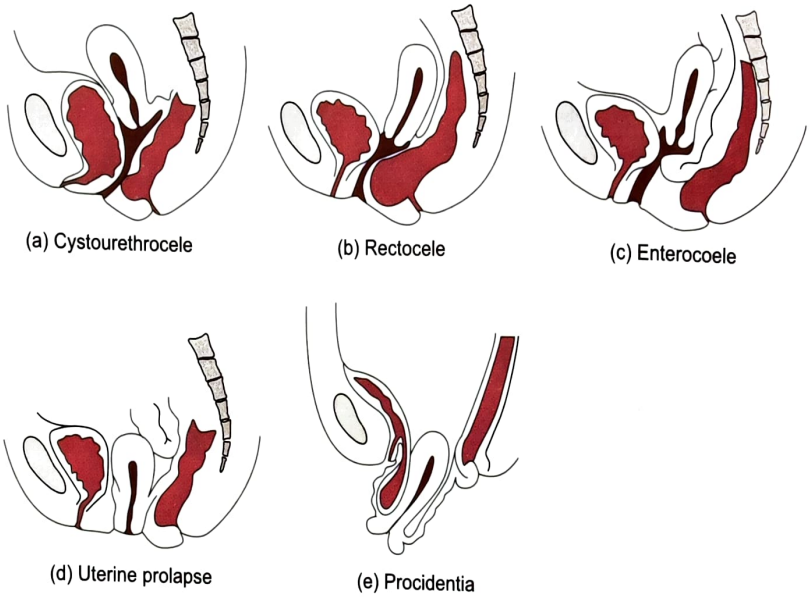


Fig. 21.1: Classification of genital prolapse

➢ ***Latest classification***

Uterine prolapse	Vaginal prolapse		
• 1 –Descent of uterus but cervix remains within the introitus • 2 –Descent to extent that the cervix projects through the vulva when woman is standing or straining • 3 –Complete procidentia	Anterior compartment Cystocele Urethrocele	Middle compartment Enterocele and massive eversion	Posterior compartment Rectocele and perineal body defect

Note: To differentiate between rectocele and enterocele, the index finger of the clinician's left hand is placed in the rectum with the tip directed upwards. Two fingers of the right hand are then placed in the vagina. The patient is asked to strain downwards. If a bulge is felt between the examining fingers in the space between the rectum and upper posterior vaginal wall, it is most likely to be an enterocele. On the other hand, if the bulge is felt on the tip of the index finger in the rectum, the bulge is most likely to be a rectocele.

Etiology

1. Postmenopausal atrophy
   a. Withdrawal of estrogen – decreased blood supply.
   b. Atrophy and atonicity of the genital supports.
2. Birth trauma
   a. Repeated delivery at short intervals.
   b. Delivery of big baby.
   c. Prolonged bearing down in the 2nd stage.
   d. Ventouse extraction of fetus before full dilatation of cervix.
   e. Crede's method to deliver placenta.
   f. Premature bearing down before full dilatation of cervix.
   g. Episiotomy not given timely.
   (all these cause overstretching of pelvic floor muscles – atonicity).
   h. Pudendal nerve injury during childbirth.
3. Genetic – congenital weakness of pelvic floor muscles.
4. In nulliparous women
   a. Spina bifida oculta.
   b. Split pelvis.
5. Increased intraabdominal pressure – chronic cough, chronic constipation.
6. Surgery
   a. Abdominoperineal excision of rectum.
   b. Radical vulvectomy.

Symptoms

1. Postural – Patient complains of something coming down or out per vagina more on standing, straining, on coughing and reduces on lying down.
2. Urinary
   a. Imperfect control of micturition – lack of support to the sphincter muscles of urethra
   b. Stress incontinence – disappears or it may be a symptom
   c. Frequent micturition – due to chronic cystitis or incomplete emptying of bladder
   d. Difficulty in micturition – patient may have to push back the cystocele vaginally to complete evacuation.
3. Rectal – difficulty in defecation and constipation (rare).
4. Discharge PV – due to chronic inflammation or bleeding of decubitus ulcer.
5. Backache – vague midsacral backache, towards end of the day without local tenderness.
6. Infertility.

➢ **Signs on per speculum examination**

1. Vaginal wall prolapse whether cystocele, rectocele, urethrocele or enterocoele. In uterovaginal the part whose supports are more defective appears first on straining.
2. Cervix – hypertrophy, cervicitis, decubitus ulcer, supravaginal elongation.
3. Vaginal walls – secondary changes. There is loss of rugosity. It is dry (exposure to air), edematous, keratinized (white), thickened due to chronic irritation. Pigmentation and decubitus ulcer may be present due to vascular stasis.
4. Uterocervical length is increased.

➢ **Investigations**

1. Blood – sugar, urea, creatinine
2. Urine examination and culture
3. X-ray chest
4. ECG
5. Pap smear
6. USG (if any pathology found then do IVP or if residual urine > 50 ml).

➢ **Differential diagnosis:**

i) Gartner's duct cyst (remnant of Wolffian duct)
- Present on anterolateral wall of vagina
- No cough impulse transmit
- Non reducible

ii) Fibroid polyp

On P/V mass present but no opening

Bimanual – Mass is fixed to cervix by peduncle.

iii) Congenital elongation of cervix
- Vaginal part up to 5 cm (but uterus in normal position).

iv) Chronic inversion of uterus.

➢ **Management**

1. Prophylaxis:

a. Antenatal – Good nutrition, antenatal physiotherapy and exercise.
b. Intrapartum
   i. Avoid premature bearing down.
   ii. Avoid premature application of forceps.
   iii. Give liberal episiotomy when required.
   iv. Suture all tears carefully.
   v. Avoid prolonged 2nd stage.
c. Postpartum
   i. Postnatal physiotherapy – perineal exercises.
   ii. Early ambulation.

iii. Adequate gap between deliveries.

2. **Non – operative treatment:**

a. Pelvic floor exercise (Kegel's exercise) – Voluntarily contracting perineal muscles as if to stop the act of defecation (15-20 times, each lasting about 5 seconds, 3-6 times a day for 3-6 months).

b. Pessary – Palliative treatment, commonly used is Ring Pessary (latex) which is available in 3 sizes – small, medium and large.

i. Indications

- During early pregnancy (T1).
- During postpartum period (up to 6 months).
- Patient not willing for surgery.
- Patient not fit for surgery.

ii. Contraindications – infection, complete prolapse, stress incontinence.

iii. Complications – infection, discharge, ulceration, dyspareunia.

[Pessary must be removed, cleaned and reinserted every 3 months).

3. **Surgical treatment –** Surgery is the definitive treatment for prolapse.

Function To Be Preserved			Operation Of Choice
Child Bearing	Menstrual	Sexual	
✓	✓	✓	Abdominal Sling operation
x	✓	✓	Fothergill's operation
x	✓	✓	Abdominal Sling operation with tubal ligation
x	x	✓	Vaginal hysterectomy with PFR
x	x	✓	Le-Fort's repair

a. Prolapse of vagina

i. Cystocele & Urethrocele – anterior colporrhaphy

ii. Rectocele – posterior perineorrhaphy

iii. Enterocele – culdoplasty, Moschowitz operation.

b. Vault prolapse

i. Colposacropexy

ii. Sacrospinous fixation

iii. Le-Fort's operation

iv. Shaw's operation.

1. **Anterior colporrhaphy:**

- Anterior vaginal wall is cut open by inverted T-incision with transverse incision in the bladder sulcus and through its mid-point or vertical incision up to urethral opening.
- Flaps are separated and line of cleavage i.e., vesico-vaginal space.
- Pubovesico-cervical ligament is divided and bladder is pushed up with a gauze.

- Vesico-vaginal fascia is tightened (using delayed absorbable sutures), excess vagina (redundant vagina) excised and vaginal wall sutured.

  (To correct stress incontinence – Kelly's suture to plicate bladder neck)

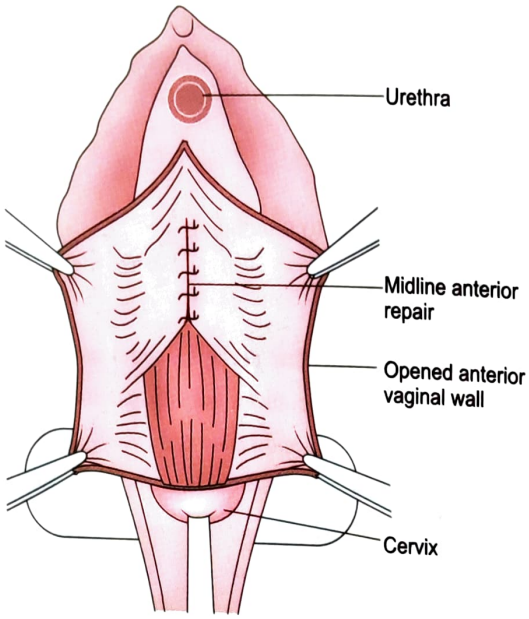


Fig. 21.2: Anterior colporrhaphy

2. **Posterior colporrhaphy / colpoperineorrhaphy:**
   - Two Allis forceps applied on the mucocutaneous junction.
   - Posterior vaginal wall cut transversely between these 2 forceps.
   - Vertical incision made from apex to the midpoint.
   - The 2 triangular flaps are dissected laterally.
   - Rectocele corrected by suturing pararectal fascia.
   - Pubococcygeous part of levator ani muscles approximated in the midline.
   - Cut margins of posterior vaginal wall approximated extending back to the perineal body and then skin.
3. **Moschowitz operation:** Abdominal repair of enterocele done by obliterating the pouch of Douglas.
4. **Fothergill's operation/ Manchester operation:**
   a. Steps:
      - Preliminary D&C – for easy passage of sutures and to remove the unhealthy endometrium.
      - Amputation of cervix.
      - Plication of Mackenrodt's (cardinal) ligaments in front of the cervix using Fothergill's stitch.
      - Anterior and posterior colporrhaphy (pelvic floor repair).

   b. Modifications:
      - Modified Manchester operation – No amputation or low amputation of cervix done in infertile patients.

- Extended Manchester operation/ Shirodkar's operation
  - * No amputation of cervix
  - * Plication of uterosacral ligament
  - * Opening of POD and high closure of peritoneum of POD
  - * Pelvic floor repair not needed.

5. **Le-Fort's operation:**
   a. Indication – very old menopausal patient with advanced prolapse who are not fit for major surgery under GA.
   b. Do Pap smear and pelvic USG to rule out pelvic pathology.
   c. Done under local anesthesia or epidural anesthesia.
   d. Steps:
      - Denudation of rectangular vaginal flap from the anterior and posterior vaginal wall.
      - Apposition of the denuded anterior and posterior vaginal walls by chromic catgut and leaving two small channels on either side for drainage.

6. **Abdominal sling operations:**
   a. Indications – Young women suffering from 2 or 3 UV prolapse who wants to retain childbearing and menstrual functions.
   b. Types:
      - ***bdominocer icope***
        - Transverse abdominal incision made through skin and fat up to rectus sheath.
        - By transverse incisions over the rectus sheath, 2 slings are elevated from the midline outwards and laterally on either side.
        - The strips are brought inside through the internal inguinal ring and sutured to the front of the cervix
      - ***P randare s cer icope***
        - Same as above procedure but the tape/strips are attached posteriorly on the cervix close to the attachment of uterosacral ligaments.
        - Other ends of the tape are brought forward retroperitoneally and attached to the external oblique aponeurosis.
      - ***Shirodkar s sling operation*** The tape is fixed posteriorly to the anterior longitudinal ligament in front of sacral promontory and anteriorly tied to supravaginal cervix on the posterior aspect.
      - ***Khanna s operation*** It is tied laterally to the periosteum of the anterior superior iliac spine and other end is fixed posteriorly on the cervix.

7. **Vaginal hysterectomy with pelvic floor repair – Ward Mayo's operation.**

Advantages	Disadvantages
- Can be done in obese patients. - Post-op complications are less. - Less mortality and morbidity.	- Experienced surgeon required. - Can't remove uterus >12 weeks.

Steps:

a. A circular incision is made over the cervix below the bladder sulcus and vaginal mucosa dissected off the cervix all around by cutting the pubovesicocervical ligament.

b. Pouch of Douglas identified and peritoneum incised.

c. Anteriorly, bladder is pushed up until UV peritoneum is visible and incised and bladder pushed forwards.

d. 1st Clamp – Mackenrodt's ligament and uterosacral ligament – clamped, cut and pedicles transfixed with chromic catgut no. 1.

e. 2nd Clamp – uterine vessels – clamp, cut and ligate.

f. 3rd Clamp – upper part of broad ligament, round ligament, fallopian tube, ligament of ovary – clamp, cut and ligate.

g. The above steps are done on either side and uterus is removed.

h. The peritoneal cavity is closed with a purse-string suture – extra-peritonisation of stump.

i. Respective pedicles are approximated in the centre.

j. Anterior colporrhaphy and posterior colpoperineorrhaphy done.

k. Vaginal packing done.

l. Self-retaining catheter introduced.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. Describe genital prolapse etiology, classification and treatment. 2. Describe the supports of uterus. D/D for mass per vagina in 35 year old lady. Describe the treatment of 3rd degree prolapse. 3. Management of III degree uterine prolapse with decubitus ulcer. 4. Enumerate causes of uterovaginal prolapse and how will you manage UV prolapse in a 25 year old woman. 5. Discuss in detail the etiopathogenesis and management of III degree prolapse. 6. What are the etiological factors for genital prolapse? Discuss the role of surgery. 7. What are the causes of mass descending per vaginum in a 30 year old woman? How will you manage genital prolapse in this patient?
Short Essays	1. Define cystocele and describe clinical features and treatment of cystocele. 2. "Decubitus ulcer". How does it occur and how will you manage it? 3. Urinary symptoms in genital prolapse. 4. Enumerate and explain the causes of urinary complications in a case of genital prolapse. 5. Management of third degree uterine prolapse with decubitus ulcer. 6. Operations for prolapse of uterus in a nulliparous woman. 7. Principles and complications of Fothergill's operation. 8. Surgery in uterine prolapse. 9. Nulliparous prolapse. 10. Cystocoele. 11. How do you differentiate enterocoele and rectocoele? 12. Fothergill's operation.

13. Pelvic floor repair.
14. Urinary symptoms in genital prolapse.
15. Signs and symptoms of prolapse of uterus.
16. Technique of vaginal hysterectomy.
17. Preoperative preparations for hysterectomy.
18. What is a decubitus ulcer? How would you treat a decubitus ulcer?
19. Etiology of prolapse of uterus.
20. What is grading of prolapse? What is the principle of treatment of nulliparous prolapse?
21. Cystocele definition, clinical features and treatment.
22. Complications of prolapse uterus.
23. Pessary treatment in prolapse indications and complications.
24. Surgery in uterine prolapse.

Short Answers

1. Classification of uterovaginal prolapse.
2. Indication(s) for rectal examination in gynecology practice.
3. Mention the differential diagnosis of mass per vagina.
4. Reason(s) for stress urinary incontinence in a patient with uterine prolapse.
5. What are the preventive measures for genital prolapse?
6. Treatment of cystocele.
7. Prevention of vault prolapse in vaginal hysterectomy.
8. Purandare's cervicopexy.
9. Components of cystocele.
10. Secondary changes in cervix in a patient with uterine prolapse. List 2 reasons.
11. Intra operative complications of hysterectomy.
12. Which are the pedicles to be cut and ligated in hysterectomy in order?
13. Etiology of genital prolapse.
14. Enterocele.
15. Decubitus ulcer.
16. Treatment of decubitus ulcer.
17. Differential diagnosis of mass per vagina.
18. Preventive measures for genital prolapse.
19. Components of Fothergill's operation.
20. Fothergill's operation.
21. Preoperative investigations for hysterectomy in a 40 year old woman.
22. Complications of vaginal hysterectomy.

22 CHAPTER

Displacements

FIXED RETROVERSION OF THE UTERUS

- **Definition**

  It is a condition in which the uterus is fixed in retroversion by adhesions or tumor.

- **Etiology**

  1. Pelvic inflammatory disease
  2. Pelvic tumors
  3. Chocolate cyst of ovary
  4. Pelvic endometriosis.

- **Clinical symptoms**

  1. Spasmodic dysmenorrhea
  2. Pelvic congestion syndrome
  3. Low backache and pelvic pain
  4. Dyspareunia.

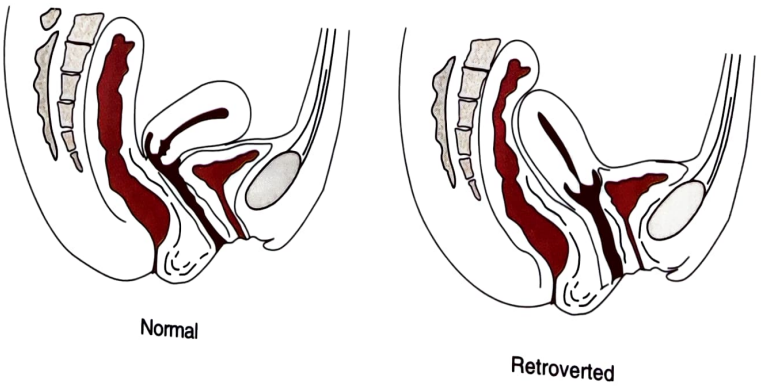


Fig. 22.1: Retroversion of the uterus

Management

a) Medical:

Pessary treatment (Hodge pessary) – The pessary puts tension on the posterior vaginal fornix and the overlying uterosacral ligaments, and this in turn holds the cervix backwards.

b) Surgical:

1. Baldy Webster operation – Round ligament loops are passed through the anterior and posterior leaves of the broad ligament stitched together behind the uterus, creating a hammock-like effect.
2. Modified Gilliam's operation – Bringing a loop of each round ligament out through the internal abdominal ring and suturing it to the back of the rectus sheath using a permanent suture.

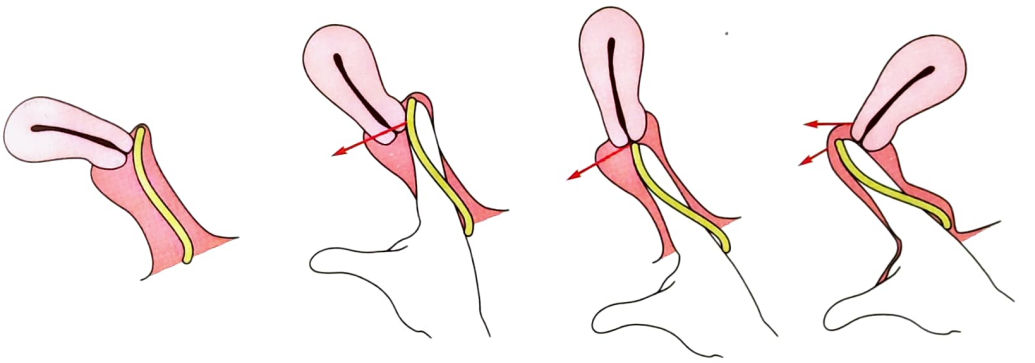


Fig. 22.2: Application of pessary for retroverted uterus

INVERSION OF THE UTERUS

Definition

Inversion is a condition in which the uterus turns inside out, the fundus prolapsing through the cervix. It is a rare complication of mismanaged labour.

Acute inversion occurs during the third stage of labor. The cervix is then open and atonic, and the fundus passes through it because of mismanagement of the third stage of labor; pressure on the fundus by the attendant; traction on the umbilical cord of an unseparated placenta. Patient presents with postpartum hemorrhage and shock.

Chronic inversion occurs when the uterus undergoes incomplete inversion at the time of delivery that the process of inversion occurs gradually during the puerperium. Hence, inversion is 'chronic' in the sense that it is only discovered a few weeks or months after delivery.

Differential diagnosiss

1. Senile inversion of uterus if the cervix has previously been amputated at a high level
2. Inversion due to pedunculated tumor arising from a stalk attached to the interior of the uterus.

PREVIOUS EXAMINATION QUESTIONS	
Short Essays	1. Causes and management of fixed retroversion of uterus. 2. Acute inversion of uterus. 3. What are the causes and differential diagnosis of uterine inversion? How will you manage puerperal uterine inversion? 4. Chronic inversion of uterus. 5. Causes of retroverted uterus.
Short Answers	1. Causes of retroverted uterus.

Fibromyomas of the Uterus

CHAPTER 23

FIBROMYOMAS

- **Definition** – it is a benign neoplasm of the female, derived from the smooth muscle rests; either from vessel wall or uterine musculature.
- **Etiology:**

  Estrogen, growth hormone and placental lactogen are important for its growth.
- **Risk factors for fibroid:**

Increased risk	Decreased risk
• Age – 35-45 years. • Nulliparous or low parity • Obesity • Early menarche < 10 years • Strong family history • DM, HTN	• Exercise • Increased parity • High intake of green vegetables • Progesterone only contraceptives • Cigarette smoking

- **Classification:**

1. Uterine
   a. Body of Uterus
      i. Submucous (5%)
         - Sessile
         - Pedunculated (becomes a 'fibroid polyp')
      ii. Intramural (interstitial) (75%)
      iii. Subserous (10%)
         - Sessile
         - Pedunculated
   b. Cervix (1-2%)
      i. Supravaginal
      ii. Vaginal.

2. Extrauterine
   a. Round ligament
   b. Broad ligament
   c. Uterosacral ligament
   d. Vagina
   e. Vulva.
3. Unusual Forms
   a. Intravenous leiomyomatosis
   b. Disseminated intraperitoneal leiomyomatosis
   c. Parasitic/ wandering fibroid
   d. Pseudocervical fibroid
   e. False broad ligament fibroid.

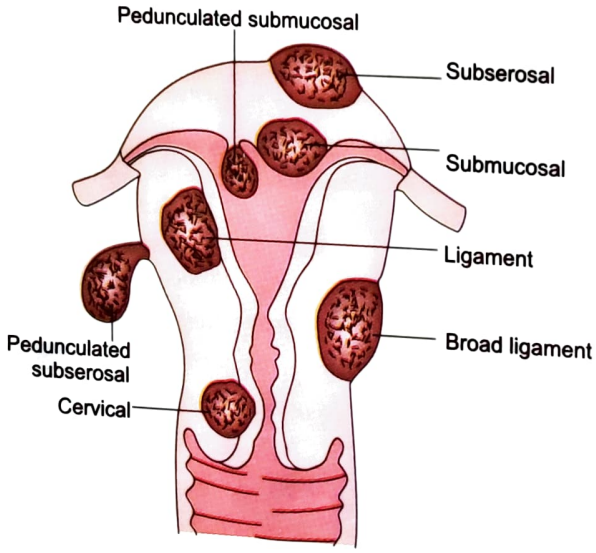


Fig. 23.1: Types of fibroids

Gross

➢ **Anatomy**

- ✓ Well circumscribed with a pseudocapsule.
- ✓ Firm in consistency.
- ✓ Cut surface – pinkish white with whorled appearance.
- ✓ Microscopy – muscle cell bundles separated by fibrous strands.
- ✓ Red degeneration – during pregnancy, becomes tense & tender with pain abdomen and fever.

Secondary changes in fibroids

1. Atrophy – Following menopause and in the postpartum period, the tumor atrophies due to decreased vascularity, shrinks in size and becomes firm and fibrotic.
2. Hyaline degeneration – Commonest type of degeneration. Usually seen in tumors more than 4 cm.The tumor loses the typical whorled appearance and its cut surface becomes smooth and homogenous. Muscle cells are replaced by hyaline material.
3. Cystic degeneration – Liquefaction of hyaline material following hyaline degeneration leads to cystic spaces in the fibroid. Tumor becomes soft due to the cystic degeneration and is asymptomatic.
4. Calcareous degeneration – There are deposits of calcium in the periphery, along the course of the blood vessels resulting in 'egg-shell' calcification; or the whole fibroid may become calcified when it is sometimes called a 'womb stone'.
5. Red degeneration – It's a degenerative change occurring during pregnancy or in the postpartum period. The exact cause is not known but it may be due to increased vascularity during pregnancy. Fibroid appears purple and thrombosis of the vessels of capsule and the tumor is usually found. Clinically there is pain and fever with marked uterine tenderness. The condition has to be differentiated from other causes of acute abdomen. Management is conservative with analgesia and maintenance of hydration.
6. Fatty change – Occurs usually after menopause and is not very common. Fat cells are found among muscle cells.
7. Sarcomatous change – Very rare. Clinically presents with rapid growth of the fibroid with irregular bleeding and acute pain in the peri or postmenopausal age group.

Complications

1. Capsular hemorrhage (rupture of surface)
2. Inversion of uterus
3. Torsion
4. Infection
5. Endometrial Ca.

Symptoms

1. Abnormal Uterine Bleeding – due to:
   - Increased surface of endometrium
   - Interference with contraction
   - Venous obstruction causing congestion
   - Endometrium – improper maturation, improper ripening & shedding
   - Increased vascularity
   - Hyperestrogenic state
   - Infection and necrosis over surface
   - Prostaglandin imbalance

2. Dysmenorrhea – congestive and spasmodic type
3. Pain – acute in torsion, hemorrhage, red degeneration, infection
4. Pressure symptoms
   - Bladder – increased frequency, retention
   - Ureter – hydroureter, hydronephrosis
   - Bowel – constipation, tenesmus
   - Pelvic veins – edema of legs
5. Lump in the abdomen
6. Vaginal discharge
7. Infertility – associated with PID, endometriosis, distortion of uterine cavity, etc
8. Recurrent pregnancy loss – especially in submucosal myoma.

➢ **Signs**

Per abdomen – Palpable mass if >12 weeks size, suprapubic, midline swelling, firm, nodular, well defined, arising from pelvis, mobile side to side, non-tender, dull on percussion.

Per vaginum – Transmitted movements – present, i.e., cervix moves when fibroid is moved, uterus not separately felt, enlarged, nodular, notch not felt.

➢ **Investigations**

a. USG – to confirm the diagnosis, number, location and size. Appears as a well-defined, hypoechoic lesion
b. Hysterosalpingography – mainly for diagnosis of submucous fibroid or polyp
c. Hysteroscopy – mainly for diagnosis of submucous fibroid or polyp
d. Diagnostic laparoscopy – to differentiate from ectopic pregnancy, PID and uterine inversion
e. MRI
f. IVP – intravenous pyelography if urinary pressure symptoms present.

➢ **Management**

Medical:

1. Progesterones – Norethisterone acetate or MPA 5-10 mg (day 5-25) – controls menorrhagia.
2. Antiprogesterone – Mifepristone (RU 486) 50 mg/day – 3 months – causes amenorrhea and shrinkage of fibroid and androgenic effect.
3. Androgens – Danazol 400 mg/day for 4 months – reduces size of tumor, reduces blood loss. Side effects - hirsutism.
4. GnRH agonists – Leuprolide IM – causes sustained pituitary down regulation and suppression of ovarian function.
5. GnRH antagonists – Cetrorelix IM – suppresses pituitary and ovaries directly.
6. Oral contraceptive pills – reduce size.
7. SERM's (Selective Estrogen Receptor Modulators).
8. SPRM's (Selective Progesterone Receptor Modulators).
9. Progesterone IUCD.
10. Aromatase inhibitors.

Surgery:

1. **Myomectomy –** it is the enucleation of the myoma(s) from the uterus leaving behind a potentially functioning organ capable of future reproduction. It is indicated in young woman who have not completed the family and presenting with symptomatic fibroids.

2. **Uterine artery embolization** – The aim of this procedure is to cause avascular necrosis and shrinkage of the fibroid by occluding the feeding artery (uterine) with polyvinyl alcohol particle introduced through percutaneous femoral artery catheterization.
3. **Hysterectomy** – It is an operative procedure to remove uterus either through an abdominal or vaginal route. It is the preferred treatment of choice in woman over 40 yrs of age.

PREVIOUS EXAMINATION QUESTIONS

Long Essays

1. Fibroid uterus classification, diagnosis and treatment.
2. Describe the complications, management of fibroid uterus.
3. A 30 year old woman presents with menorrhagia and menstrual abnormality. List the different diagnosis. How will you do diagnose and manage myoma uterus?
4. Describe the clinical features and management of fibroid uterus in a twenty five year old lady.
5. What are the causes of menorrhagia in a 35 year old woman and how will you manage a case of fibroid uterus in such a woman?
6. Describe the clinical features and management of fibroid uterus in a twenty year old lady.
7. A 30 year old infertile woman has 16 week size uterus with multiple fibroids. Describe the management.
8. Discuss the etiology of fibroid uterus and the different modalities of treatment of fibroid uterus.
9. What are the clinical features of fibroid uterus? How will you investigate and manage a case of fibroid uterus?
10. Enumerate the differential diagnosis of mass in lower abdomen. How would you treat 20 weeks of fibroid with menorrhagia at the age of 42 years?
11. Describe the various indications for abdominal hysterectomy and in detail discuss the complications.

Short Essays

1. Types of fibroid uterus and their clinical features.
2. Clinical features and management of red degeneration of uterine fibroid.
3. Fibroid polyp clinical features and treatment.
4. What are the clinical features and management of submucous fibroid?
5. Causes and types of abnormal uterine bleeding in a patient with fibroid uterus.
6. Management of fibroid uterus detected in woman aged 26 years.
7. Medical management of fibroid uterus.
8. 10 indications for hysterectomy.
9. Abdominal hysterectomy.
10. Fibroid polyp.
11. Submucous fibroid.
12. Principles of myomectomy.
13. Myoma uterus–complications.

	14. Management of fibroid uterus detected in woman aged 26 years. 15. Mention prerequisites of myomectomy. 16. Write the indications, procedure and complications of myomectomy. 17. Complications of fibroids. 18. Symptoms of fibroid uterus. 19. What are the clinical features and management of submucous fibroid? 20. Myomectomy. 21. Broad ligament fibroid.
Short Answers	1. Day wise postoperative management after abdominal hysterectomy. 2. Indications for hysterectomy. 3. Predisposing features for fibroids. 4. Fibroid polyp. 5. What are the types of fibroids? 6. Complications of fibroid uterus. 7. Red degeneration of fibroid. 8. Cause(s) of abdominal/ pelvic pain in fibroid uterus. 9. Fibroid polyp–diagnosis and treatment. 10. Causes of menstrual problems in fibroid uterus. 11. Indications for surgery in fibroid uterus. 12. Mention prerequisites for myomectomy. 13. Mention the indications for vaginal hysterectomy.

Disorders of the Ovary

CHAPTER 24

FUNCTIONAL CYSTS OF THE OVARY

- Functional cysts are a result of accumulation of fluid within the functional unit of the ovary.
- These are recognized from neoplastic cysts by the fact that:
  1. Never grow more than 7 cm in size
  2. Unilocular with clear fluid
  3. Regress after some time.
- These include:
  1. Follicular cysts
  2. Lutein cysts
  3. PCOS/PCOD.

FOLLICULAR CYSTS

- They may be single or multiple.
- Generally do not exceed 5 cm in diameter.
- They are result of failure of absorption of the fluid in an incompletely developed follicle or anovulation.
- Usually asymptomatic unless hemorrhage, rupture or torsion supervenes.
- Large and multiple cysts may cause pelvic pain, dyspareunia, and irregular bleeding.
- Treatment – oral medroxyprogesterone 10 mg tid over a period of 5-7 days.

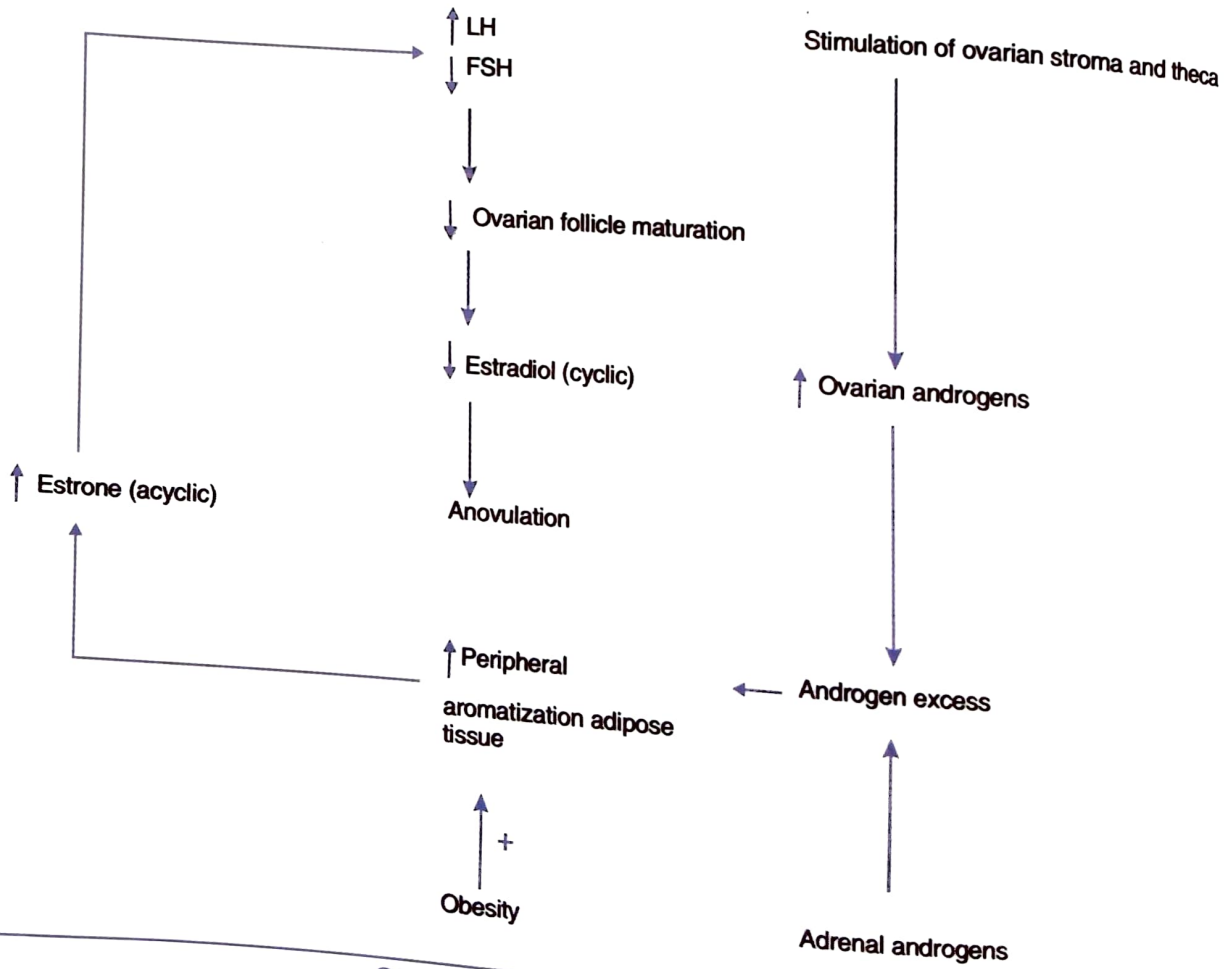
LUTEIN CYSTS

- Caused due to excessive chorionic gonadotropin secreted in cases of gestational trophoblastic tumors.
- Types – Two types are recognized:
  1. Granulosa lutein cysts
  2. Theca lutein cysts

- **Granulosa Lutein (Corpus Luteum) Cysts** – lined by granulosa lutein cells
  1. Found within the corpus luteum
  2. Clinical features - may cause local pain, tenderness or delayed menstruation and are often palpable clinically
  3. Complications - torsion or rupture
  4. Treatment - most will resolve in due course of time. Complications lead to an acute abdomen requiring surgical treatment.
- **Theca Lutein Cysts** – lined by theca lutein cells
  1. Usually bilateral
  2. Filled with straw colored fluid
  3. Often associated with hydatidiform mole, choriocarcinoma and hCG or clomiphene therapy
  4. Spontaneously regress after elimination of the mole, therapeutic curettage, destruction of choriocarcinoma or discontinuation of hCG therapy.

POLYCYSTIC OVARIAN DISEASE (PCOD) [Stein-Leventhal syndrome]

- **Definition** - PCOD is a state of androgen excess and chronic anovulation associated with normal or raised estrogen, raised LH and low FSH:LH ratio.



- **Patient Profile**
  1. Age incidence - 15-25 years of age
  2. She complains of oligomenorrhea and often amenorrhea of a few months duration
  3. Infertility occurs in 30% cases
  4. Obesity and hirsutism are the additional features.
- Fault lies with the ovary itself, but may be also related to HPO axis and adrenal gland (hyperandrogenemia). Increased E2 levels results in a negative feedback on the pituitary resulting in decreased levels of FSH and LH.
- **Macroscopic Features**
  1. Ovaries are often bilaterally enlarged with thick capsule
  2. Surface may be lobulated
  3. Peritoneal surface is free of adhesions
  4. Multiple cysts 0.5-1 mm (at times upto 20 mm) in size are localized along the surface of the ovary giving a "necklace" appearance on ultrasound. These are atretic follicles
  5. Theca cell hyperplasia is seen which produces excess testosterone secretion.

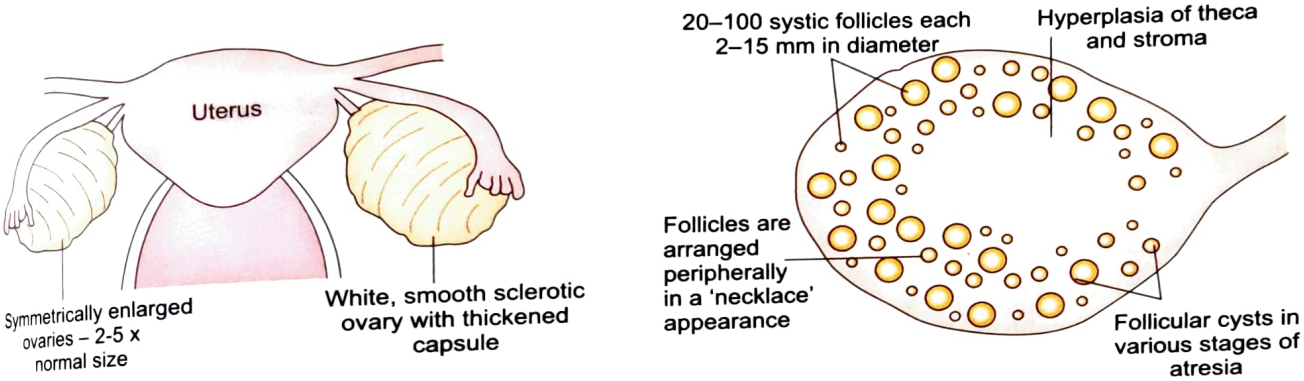

Uterus
Symmetrically enlarged ovaries – 2-5 x normal size
White, smooth sclerotic ovary with thickened capsule
20–100 systic follicles each 2–15 mm in diameter
Hyperplasia of theca and stroma
Follicles are arranged peripherally in a 'necklace' appearance
Follicular cysts in various stages of atresia

Fig. 24.1: Appearance of ovary in PCOD

- **Differential diagnosis**
  1. Congenital adrenal hyperplasia
  2. Cushing's syndrome
  3. Androgen secreting tumor.
- **Investigations**
  1. Ultrasound shows several subcapsular cysts of varying size, which is diagnostic
  2. Diagnostic laparoscopy
  3. Low FSH:LH ratio
  4. Raised testosterone, androstenedione and DHEA.
- **Treatment**
  1. General Therapy
     a. Weight loss of more than 5% of previous weight

b. Avoidance of cigarette smoking.

2. Medical Therapy
   a. Estrogen – suppresses androgen and adrenal production. It is best given cyclically with progestogen with no androgenic properties
   b. Dexamethasone, prednisone – reduces androgen production
   c. Hirsutism – treated with cyproterone acetate or spironolactone
   d. Infertility – treated with clomiphene.
3. Surgical Therapy
   a. Indications:
      1) Medical therapy fails
      2) Hyperstimulation occurs
      3) Use of GnRH analogues is unaffordable.
   b. Types:
      1) Laparoscopic multiple punctures of the cysts with electrocautery or laser
      2) Wedge resection is now obsolete due to post-op complications.

OVARIAN TUMORS

Based on their nature, they are classified as benign or malignant; further sub- classified based on cell of origin.

WHO Classification of ovarian tumors (Based on cell of origin)

1. Epithelial Tumors
   a. Serous tumors
   b. Mucinous tumors
   c. Endometrioid tumors
   d. Clear cell (mesonephroid) tumors
   e. Brenner tumors
   f. Mixed epithelial tumors
   g. Undifferentiated carcinoma
   h. Unclassified epithelial tumors.
2. Sex Cord Stromal Tumors
   a. Granulosa cell tumors, theca cell tumors
   b. Androblastomas (Sertoli-Leydig cell tumors)
   c. Gynandroblastomas
   d. Unclassified.
3. Lipid Cell Tumors.
4. Germ Cell Tumors
   a. Dysgerminoma
   b. Endodermal sinus tumor
   c. Embryonal carcinoma
   d. Polyembryoma

e. Choriocarcinoma
f. Teratoma
g. Mixed tumors.
5. Gonadoblastoma.
6. Soft tissue tumors (not specific to ovary).
7. Unclassified tumors.
8. Metastatic tumors.

BENIGN OVARIAN TUMORS

EPITHELIAL TUMORS

1. **Serous cystadenomas**
   - Most common benign epithelial tumor
   - Bilateral in 10%
   - Usually unilocular with papilliferous processes on the inner surface
   - Occasionally have concentric psammoma bodies (intracellular calcification).
2. **Mucinous cystadenomas**
   - This is the second most common benign epithelial tumor
   - It is a large, unilateral, multilocular cyst which may reach the size of a full term gravid uterus filled with thick gelatinous fluid
   - Pseudomyxoma peritonei – Well differentiated mucinous cystadenocarcinomas or borderline tumours can result in seedling growths in the peritoneal cavity secreting mucin causing matting and obstruction of the bowel loops.
3. **Brenner tumors**
   - More common in women > 40 yrs, half of them occur as incidental findings
   - Majority are < 2 cm
   - Origin – from wolffian metaplasia of the surface epithelium
   - Microscopically– consist of islands of transitional epithelium (Walthard rests) in a dense fibrotic stroma.
4. **Endometroid cystadenoma –** These are difficult to differentiate from endometriosis.

SEX CORD STROMAL TUMORS

- Represent 4% of benign ovarian tumors.
- Can be seen at any age including children and postmenopausal women.
- They are hormone secreting and of four histological types – theca cell, granulosa cell, fibromas and Sertoli-Leydig cell tumors.

1. Granulosa cell tumors are all malignant tumors, are slow growing and largely solid. The pathognomic features are the Call-Exner bodies.
2. Theca cell tumors – almost all are benign, solid and unilateral
   - Present at 60-70 yrs
   - Estrogen secreting and can cause precocious puberty, postmenopausal bleeding, endometrial hyperplasia or cancer.
3. Fibromas – they are derived from stromal cells of the ovary

- Present around 50 yrs of age
- Grossly are hard, mobile with a glistening white surface
- Bilateral in < 10%
- Are mostly associated with ascites
- Meig's syndrome is a syndrome in which fibromas are associated with ascites and pleural effusion; is seen in 1% of fibromas.

4. **Sertoli- Leydig cell tumors –** They are commonly androgen secreting tumors causing virilization
   - They are of low grade malignancy
   - They are usually seen at about 30 yrs of age
   - Usually small and bilateral.

➢ **GERM CELL TUMORS**

➢ **Teratoma –** All germ cell tumors show differentiation along embryonic rather than extraembryonic pathways. These are grouped together as teratomas and are divided into three categories:

1. Mature (benign) - e.g., dermoid cyst
2. Immature (essential malignant) – e.g., solid teratoma
3. Monodermal(highly specialized) – e.g., struma ovarii.

➢ **Dermoid Cyst**

- Unilocular, with smooth surface, rarely more than 15 cm in diameter.
- Contains sebaceous material and hair.
- Wall is lined in part by squamous epithelium, which contains hair follicles and sebaceous glands. Teeth, bone, cartilage, thyroid tissue and bronchial mucus membrane are often found in the wall.
- Sometimes, the sebaceous material collects together in the form of small balls, and as many as 1000 sebaceous balls have been counted in a dermoid cyst.
- Inner surface is called a 'focus' or 'embryonic node' from which the hair project, and in which the teeth and bone are usually found.
- Frequently associated with mucinous cystadenomas to form a combined tumor.
- Multiple dermoid cysts in the same ovary are well recognized and it is not uncommon to find 2-3 separate dermoids.
- Extra- ovarian dermoids arise occasionally in the lumbar region, in the uterovesical area, in the parasacral region and in the rectovaginal septum.
- Dermoid cysts are innocent ovarian tumors but epidermoid carcinoma and sarcomatous changes may occur on rare occasion.

➢ **Clinical features of benign ovarian tumors**

1. Age
   a. Benign tumors predominantly manifest in the late childbearing period
   b. Dermoid (90%), especially with mucinous cystadenoma is common in the reproductive period
   c. As such, the dermoid is more common during pregnancy (10%).
2. Parity – no correlation with parity of the patient.
3. Symptoms
   a. Most tumors are asymptomatic
   b. Heaviness in lower abdomen

c. Dull aching pain in lower abdomen
d. Gradually increasing mass in lower abdomen
e. In neglected cases, tumor may be big enough to fill whole of the abdomen. It causes cardio-respiratory embarrassment or gastrointestinal symptoms like nausea or indigestion
f. Menstrual pattern remains unaffected unless associated with hormone producing tumours.

4. Signs
a. General Examination
   1) General condition remains unaffected
   2) Pitting edema of legs may be present when a huge tumor presses on the great veins.
b. Abdominal Examination
   1) Inspection:
      a) Bulging of the lower abdomen over which the abdominal wall moves freely with respiration.
      b) Mass may be placed centrally or on one side.
      c) At times, the mass fills the entire abdominal cavity everting the umbilicus with visible veins under the skin.
      d) Flanks remain flat.
   2) Palpation:
      a) Feel is cystic or tense cystic.
      b) Freely mobile from side to side but restricted from above down unless the pedicle is long.
      c) Large size or adhesions make its mobility restricted.
      d) Upper and lateral borders are well-defined but the lower pole is difficult to reach (suggestive of pelvic origin).
      e) Surface over the tumor is smooth but often grooved in lobulated tumor.
      f) Usually non-tender.
   3) Percussion:
      a) Percussion note is dull in the center and resonant in the flanks.
      b) Fluid thrill may be elicited when walls are thin and content is watery.
      c) Co-existing ascites may be present in Meigs' syndrome.
   4) Auscultation:
      a) Friction rub may be present over the tumor.
      b) Hissing sound over a vascular fibroid.
      c) Gurgling sound in ascites.
      d) FHS over a pregnant uterus.
c. Bimanual Examination
   1) Uterus is separate from the mass, pushed to one side
   2) Groove is felt between the uterus and the mass
   3) Movement of the mass per abdomen fails to move the cervix
   4) On elevation of the mass per abdomen, cervix remains stationary
   5) Lower pole of the cyst can be felt through the fornix
   6) Absence of pulsation of the uterine vessels through the fornices.

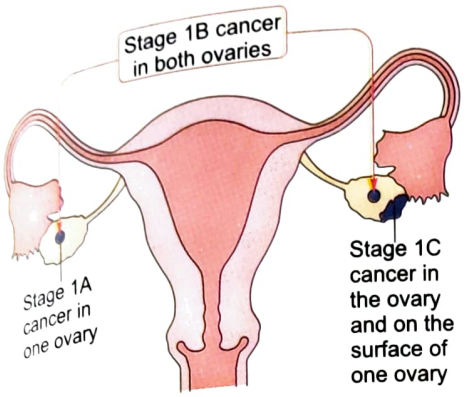
- **Complications**
  1. Torsion of the pedicle (axial rotation) → congestion → hemorrhage
  2. Rupture (traumatic/ spontaneous)
  3. Intracystic hemorrhage
  4. Infection
  5. Pseudomyxoma peritonei
  6. Malignancy.
- **Investigations**
  1. Ultrasound – It can identify the uterus and the tumor in the same scan
  2. Straight X-ray – Finding of a shadow of teeth or bones is direct evidence of a dermoid cyst. An outline of a soft tissue shadow may also be visible
  3. IVP exclude hydronephrosis
  4. Laparoscopy – to differentiate painful cystic mass from disturbed ectopic
  5. Laparotomy – specially indicated when a suspected functional cyst fails to regress in follow-up period
  6. Cytology – When the patient presents with ascites or pleural effusion, cytological examination of the aspirated fluid is done for malignant cells
  7. CT, MRI, Doppler
  8. Tumor markers – CA-125, hCG, AFP, LDH.
- **Treatment –** Laparotomy is necessary in every case, not only for obtaining the specimen for histology by removal of the tumor, but also to stage and carry out the definitive debulking operation. The treatment comprises:
  1. Abdominal hysterectomy with BSO
  2. Ovariotomy
  3. Cystectomy
  4. Laparoscopic ovariotomy
  5. Laparoscopic cystectomy.

MALIGNANT OVARIAN TUMORS

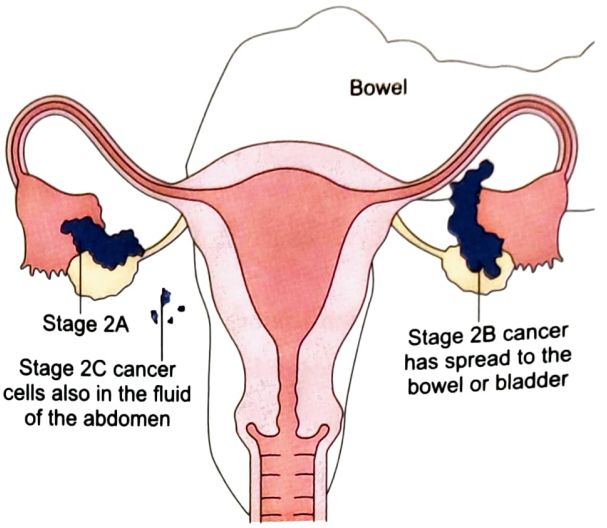
- **Risk factors**
  - Early menarche, late menopause and nulliparity are associated with an increased risk due to the theory of 'incessant ovulation" causing repeated epithelial damage and reconstruction. Use of oral contraceptive pills and pregnancy suppress ovulation and decrease the risk.
  - Infertility treatment – prolonged treatment with ovulation inducing agents increases the risk.
  - Family history – inheritance plays a role in 5% of epithelial ovarian cancers.
- **Classification** is as shown in WHO classification.
- **Spread of ovarian cancer**

  Ovarian cancer spreads by direct, lymphatic and blood spread.
  - Direct spread occurs to other pelvic and intraabdominal organs, the omentum and under surface of the diaphragm.
  - Lymphatic spread occurs along the lymphatics running along with the ovarian vessel to the para-aortic lymph nodes at the level of the renal vessels. Spread to the mediastinal and cervical lymph nodes occurs from the para-aortic nodes. Inguinal lymph nodes may be involved by retrograde spread via the ovarian ligaments.
  - Blood spread occurs to the liver, lung and occasionally to the bone and brain.

Staging



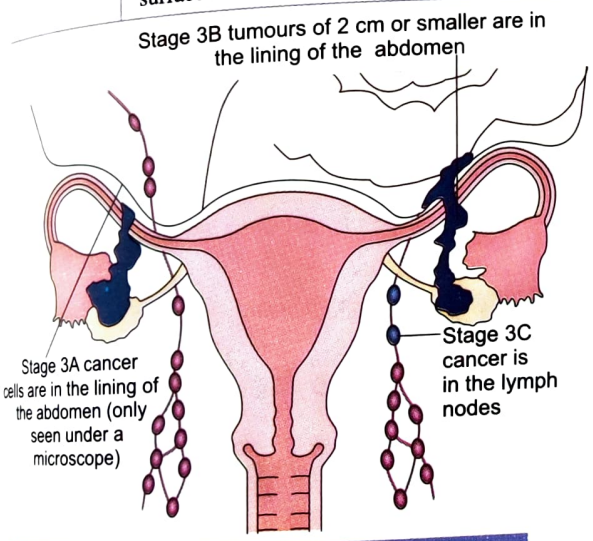
(a)



(b)

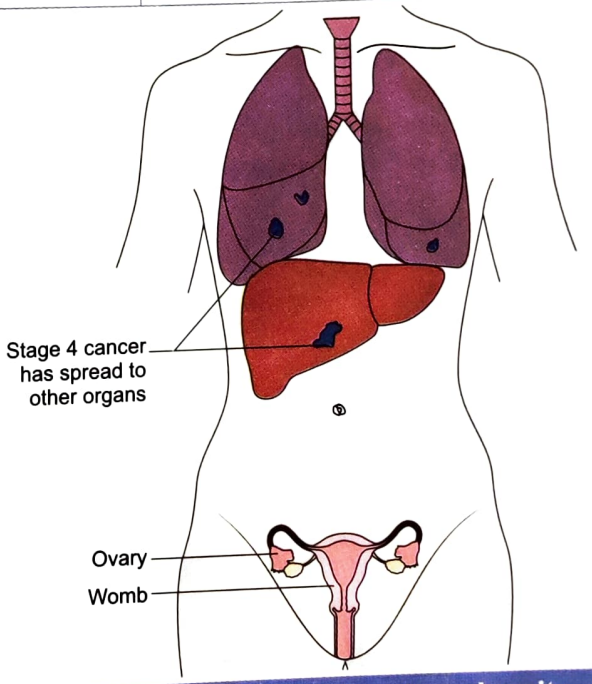
Stage I	Confined to Ovaries
IA	1 Ovary
IB	2 Ovaries
IC	1/2 + Capsule rupture / Tumor at surface / Malignant ascites

Stage II	1/2 Ovaries + Pelvic Extension
IIA	Uterus / Tubes
IIB	Other pelvic organs
IIC	A/B with malignant ascites


Stage 3B tumours of 2 cm or smaller are in the lining of the abdomen
Stage 3A cancer cells are in the lining of the abdomen (only seen under a microscope)
Stage 3C cancer is in the lymph nodes

Stage III	1/2 Ovaries + Peritoneal Mets/ Reg LNs
IIIA	Micro peritoneal mets
IIIB	Macro < 2 cm peritoneal mets
IIIC	Macro > 2 cm/Reg LNs mets

(c)



Stage IV	Distant mets beyond peritoneal cavity

(d)

Fig. 24.2: Staging of malignant ovarian tumors

Staging laparotomy is usually combined with the definitive surgery. To assess the extent of the disease locally, at the time of laparotomy, cytologic washings of the upper abdomen and the diaphragm, infracolic omentectomy, pelvic and para aortic lymph nodal sampling and random peritoneal biopsies are also performed.

➢ **Management**

a. Conservative management

Simple ovarian cysts <8 cm in diameter with normal CA-125 should be observed and reexamined after 3-6 months.

b. Surgery for ovarian tumors

1. Surgery is indicated if the cyst significantly increases in size.
2. The aims of surgery are:
   - Diagnostic to know the nature of the tumor and to stage the disease
   - Therapeutic to excise benign lesions and debulk the malignant ones.
3. The surgical options are:
   - Women <35 yrs
     - Unilateral tumor (benign appearing) Ovarian cystectomy (excision of the cyst from the ovary) or salpingo-oophorectomy (removal of the tube and ovary), should be carried out.
     - If suspicious of malignancy unilateral salpingo-oophorectomy is carried out and the peritoneal washings are taken for cytology. If the cytology is positive, chemotherapy is given in the postoperative period.
   - Women 35-40 yrs treatment should be individualised based on the completeness of her family and the appearance of the tumor at surgery. Benign appearing ovarian cystectomy or salpingo-oophorectomy.
     - If her family is complete, total abdominal hysterectomy with bilateral salpingo-oophorectomy with omentectomy is carried out, along with peritoneal washings for cytology.
     - If her family is not complete conservative surgery can be carried out after thorough staging and counselling regarding the risk of recurrence.
   - Advanced stage ovarian cancer maximal cytoreductive surgery should be the aim to reduce the volume of residual disease to <1 cm.
   - Interventional debulking surgery A biopsy is taken at initial surgery followed by chemotherapy and repeat surgery. It is still undergoing clinical trials at present.
   - Second-look surgery planned laparotomy at the end of chemotherapy has not been shown to improve survival and is not recommended routinely.
   - Laparoscopic surgery The indications are for diagnosis, when the nature of the pelvic mass is uncertain or for the management of a benign simple ovarian cyst.

c. Adjuvant chemotherapy

Patients with residual epithelial ovarian cancer of >1 cm following surgery and patients stage IC and higher, need adjuvant therapy to halt progression and prevent a relapse. The agents used are carboplatin + paclitaxel (CP) or cisplatin + doxorubicin + cyclophosphamide (CAP) are given parenterally every three weekly for 6 cycles.

Second Look Surgery

A second look procedure is a complete surgical exploration and restaging procedure to determine the status of the cancer upon completion of chemotherapy. In many cases, after an ovarian cancer has been diagnosed and treated, there is no detectable cancer. X-rays, CT scans, CA-125 and physical examination are all normal. The question is then asked "What to do now?". If there is no more cancer then nothing more needs to be done. If there is still cancer remaining in the abdomen, more treatment needs to be given. At this point the only way to determine the status of the cancer is by another major surgery, because even though all the tests are negative there can still be cancer present. This is called a second look surgery.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. Discuss diagnosis and management of polycystic ovarian syndrome. 2. Classify ovarian tumors. Mention its complications and management in postmenopausal woman. 3. Different modalities of diagnosis and management of malignant serous cystadenocarcinoma. 4. Describe the diagnosis, management of PCOS. 5. Describe the benign ovarian tumors–classification, diagnosis and treatment. 6. Describe the malignant ovarian tumors–classifications, diagnosis and treatment. 7. Classify epithelial ovarian tumors. Describe the clinical features of benign ovarian tumours and management of serous cystadenoma in a 30 year old lady. 8. Classify germ cell tumors, clinical features, diagnosis, management of any one of them in a young woman. 9. Discuss the diagnosis and management of malignant ovarian tumour. 10. Classify ovarian tumors. Write the diagnosis and management of benign cystic teratoma. 11. Classify ovarian tumors. Discuss the principles of management of malignant ovarian tumors. 12. Stage (FIGO) ovarian neoplasm. Add a note on treatment of bilateral dermoid cyst in a 25 years lady.
Short Essays	1. Stein-Leventhal syndrome. 2. Functional cysts of the ovary. 3. Classify germ cell tumors of ovary and indicate tumour markers. 4. Dermoid cyst. 5. Cystic teratoma. 6. Dysgerminoma. 7. Feminising tumors of ovary. 8. Chocolate cyst. 9. Ovarian tumors–complications. 10. Twisted ovarian cyst. 11. Torsion ovarian cyst.

	12. Mieg's syndrome. 13. Mucinous tumors of ovary. 14. Benign ovarian cysts. 15. Differential diagnosis for mass felt through right fornix. 16. Features of malignancy in ovarian tumors on clinical examination and at laparotomy/scopy. 17. How will you diagnose and treat dermoid cyst of ovary? 18. Complications of dermoid cyst. 19. Clinical features, diagnosis and management of torsion of ovarian cyst. 20. Second look surgery in cancer ovary. 21. Mention the benign ovarian tumors. How will you manage a case of dermoid cyst of the ovary in 26 yr old woman? 22. Polycystic ovarian syndrome – diagnosis and management. 23. Postmenopausal palpable ovary. 24. CA 125–indications in gynecology. 25. Discuss the complications of benign ovarian cyst. How will you diagnose and treat dermoid cyst? 26. Give the histological features of borderline tumor of ovary. 27. Classify ovarian tumors. 28. Brenner tumor. 29. Differential diagnosis of benign ovarian tumor. 30. Complications of ovarian cysts. 31. Staging laparotomy for gynecological malignancies.
Short Answers	1. Mention the germ cell tumors of ovary. 2. Diagnosis of polycystic ovarian syndrome. 3. Name hormone producing ovarian tumors. 4. Sonographic features of malignancy in ovarian tumors. 5. Clinical presentation of benign cystic teratoma of ovary. 6. Follicular cysts in a young lady. 7. Mention the conditions where the lutein cysts are found. 8. Theca lutein cysts. 9. Write on corpus luteal cyst of ovary. 10. Write briefly on polycystic ovaries. 11. How will you diagnose PCOD? 12. Dermoid cyst of the ovary. 13. Brenner tumor. 14. Dysgerminoma. 15. Structures in the pedicles of ovarian torsion. 16. Pseudomyxoma peritonei causes, presentation. 17. Malignant features of ovarian tumor. 18. Name tumor markers in ovarian malignancy. 19. Carcinoembryonic antigen-125.

Gynecologic Oncology

CHAPTER 25

BARTHOLIN'S CYST

- **Etiology** – It may be caused by infection or trauma followed by fibrosis and occlusion of the lumen. Lateral or ill-directed mediolateral episiotomy may cause trauma.
- **Clinical Features**
  1. Small cyst often remains unnoticed.
  2. If it becomes large (size of hen's egg), there is local discomfort and dyspareunia.
  3. On examination:
     a. Unilateral swelling on the posterior half of the labia majora which opens up the posterior end of the labia minora.
     b. Its medial projection makes the vulval cleft S-shaped.
     c. Overlying skin is thin and shiny.
     d. Cyst is fluctuant and non-tender.
- **Treatment**
  1. Excision
  2. Marsupialization – The advantages of marsupialization over excision are:
     a. Simple
     b. Can be done under LA
     c. Shorter hospital stay (24 hours)
     d. Almost no post-op complications
     e. Gland function (lubricant) remains intact
  3. Fistulization using a Word catheter.

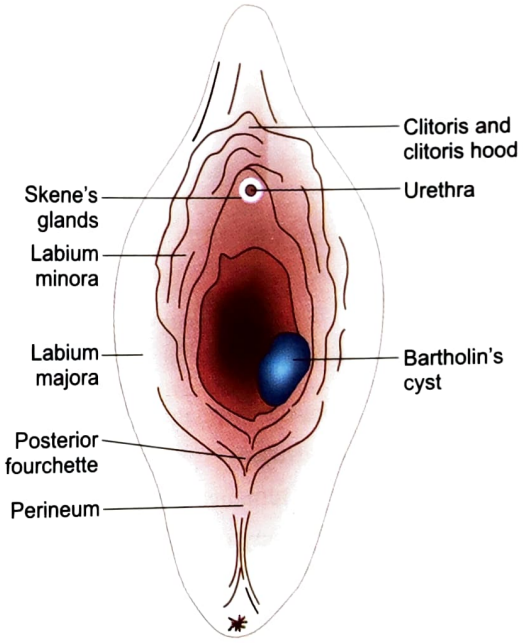


Fig. 25.1: Bartholin's cst

- **Bartholin's abscess:** A Bartholin gland abscess develops when a Bartholin cyst becomes infected. Escherichia coli is the single most common pathogen. Patient presents with a tender lump on the side of vaginal opening with fever and dyspareunia.It is treated with antibiotics before doing any of the procedure mentioned above.

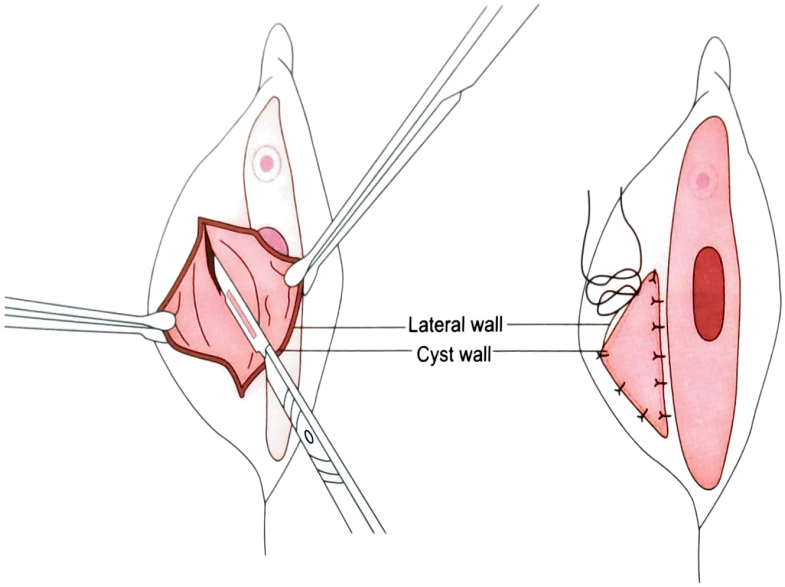


Fig. 25.2: Incision, drainage and marsupialization of Bartholin's abscess

GARTNER'S DUCT CYST

- It arises from the remnants of the mesonephric duct and lies in the anterolateral aspect of the vaginal wall.
- **Clinical Features**
  1. Small cyst remains asymptomatic
  2. Large cyst may cause dyspareunia.
- **Treatment** – Excision.

KRUKENBERG TUMOR

- Secondary growth in the ovary, from a primary carcinoma of the stomach (70%), large bowel (15%) and breast (6%)
- Almost always arise by retrograde lymphatic spread
- Almost always bilateral
- Smooth surface which may be slightly bossed
- Freely mobile in the pelvis
- No tendency to form adhesions with neighbouring viscera
- No infiltration through the capsule
- Retains the shape of the normal ovary
- Has peculiar solid waxy consistency although cystic spaces due to degeneration of the growth are common.

CARCINOMA CERVIX

Risk Factors

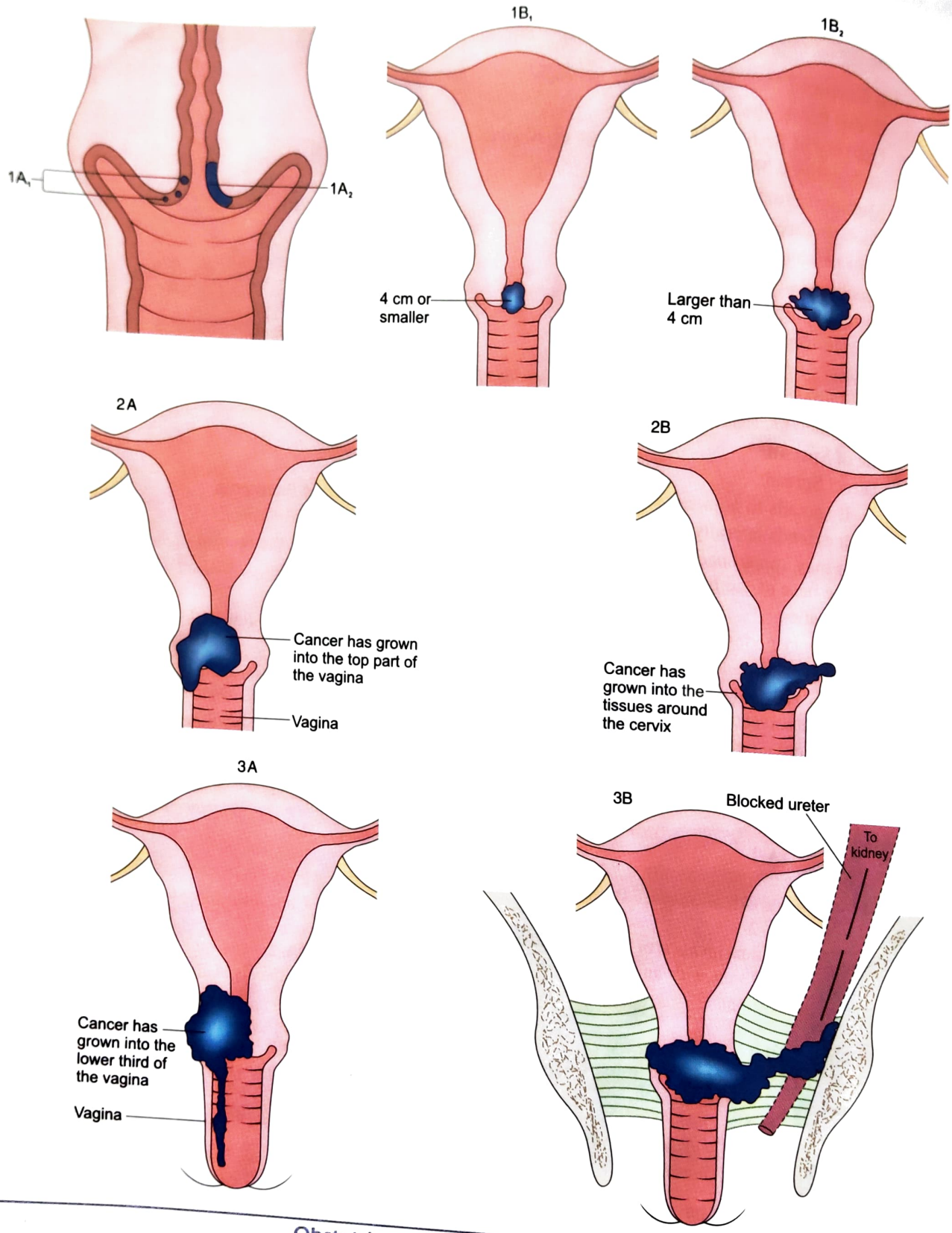
1. Coitus before the age of 18 years
2. Delivery of first baby before the age of 20 years
3. Multiparity with poor birth spacing
4. Multiple sexual partners
5. Poor socioeconomic status
6. Poor personal hygiene
7. Smoking, alcohol, drug abuse
8. Sexually transmitted diseases
9. Immunosuppression
10. History of preinvasive lesions
11. Viral infections caused by HS-2 and HPV- 16, 18, 31 & 32
12. Combined OCP and progestogen use over long periods.

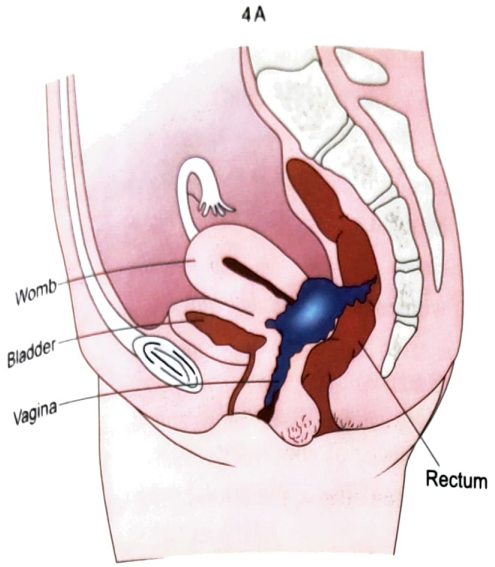
Mode of Spread

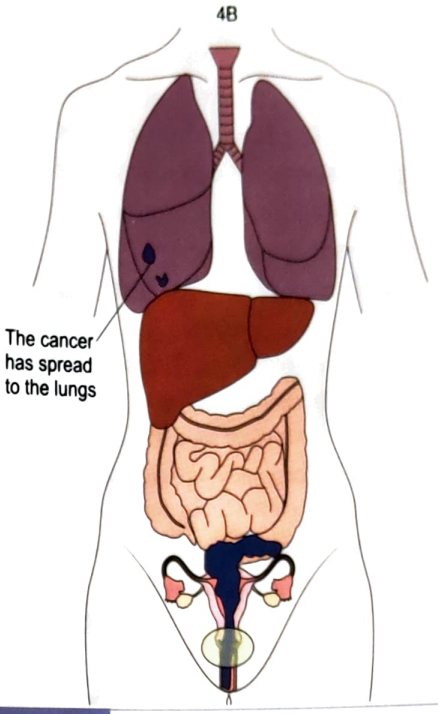
1. Direct Spread:
   a. It spreads to adjacent structures, to the vagina and to the body of the uterus.
   b. It extends laterally to the parametrium, paracervical and paravaginal tissues.
   c. It may spread backwards along the uterosacral ligament to involve the rectum or forwards to involve the base of the bladder.
2. Lymphatics
   a. Primary nodes involved – parametrial, obturator, internal iliac, external iliac and sacral
   b. Secondary nodes involved – common iliac, inguinal and para-aortic.
3. Blood – Hematogenous spread is late and usually by veins rather than arteries. Lungs, liver or bone are usually involved.
4. Direct implantation – Direct implantation of the cancer cells at operation on the vault of the vagina or abdominal or perineal wound is very rare.

Staging (FIGO)

Stage 1 The cancer is confined to the cervix	**1A-1** Stromal invasion of < 3.0 mm in depth and extension of < 7.0 mm **1A-2** Stromal invasion of >3.0 mm and not >5.0 mm with an extension of not >7.0 mm.	**Stage 2** The cancer invades beyond the cervix and involves either the upper 1/3rd of the vagina or the parametria.	**2A** Involves the upper 2/3rd of the vagina. **2A-1** Clinically visible lesion < 4.0 cm in size. **2A-2** Clinically visible lesion > 4.0 cm in size.
	1B-1 Clinically visible lesion < 4.0 cm in greatest dimension. **1B-2** Clinically visible lesion > 4.0 cm in greatest dimension.		**2B** With obvious parametrial invasion.


1A1
1A2
1B1
4 cm or smaller
1B2
Larger than 4 cm
2A
Cancer has grown into the top part of the vagina
Vagina
2B
Cancer has grown into the tissues around the cervix
3A
Cancer has grown into the lower third of the vagina
Vagina
3B
Blocked ureter
To kidney





Stage 3 The cancer extends to the pelvic wall and/or involves the lower third of the vagina and/or causes hydronephrosis.	**3A** Involves the lower third of the vagina.	**Stage 4** The cancer involves the bladder or rectum or has extended beyond the true pelvis.	**4A** Spread of the growth to adjacent organs.
	3B Extension to the pelvic wall and/or hydronephrosis.		**4B** Spread to distant organs.

Fig. 25.3: FIGO staging of Ca cervix

Clinical Features

1. Many are asymptomatic.
2. Some complain of postcoital bleeding or discharge.
3. On inspection, cervix often appears normal or there may be cervicitis or an erosion which bleeds on touch.
4. Some present with postmenopausal bleeding.
5. Cardinal signs of Ca cervix.
   - Bleeding of cervix on touch
   - Fixity of cervix due to infiltration
   - Friability
   - Induration.

Complications

1. Attacks of ureteric pain, pyelitis and pyelonephritis due to hydronephrosis
2. Pyometra
3. Vesicovaginal fistula
4. Rectovaginal fistula.

➢ **Causes of Death**

1. Uremia – due to ureteric obstruction following parametrial involvement
2. Hemorrhage – vaginal bleeding from the growth may be brisk or continuous
3. Sepsis – localized pelvic or generalized peritonitis
4. Cachexia – due to cumulative effect of the above factors
5. Metastasis – distant organs, especially lungs may be involved.

➢ **Investigations**

1. Pap smear (screening test) – should be offered to all women above the age of 18 years who are sexually active.
2. DNA study – aneuploidy is a hallmark of malignant potential.
3. Colposcopy – allows accurate delineation of suspicious areas for tissue biopsy. Suspicious areas reveal acetowhite areas, a mosaic pattern, punctation and/or presence of abnormal vessels.
4. Cervicography – photograph of the entire external os is taken after application of 5% acetic acid and sent to the colposcopist for selecting areas for biopsy.
5. Cone biopsy – both diagnostic and therapeutic.

   (Visual inspection with Lugol's iodine (VILI), also known as Schiller's test, uses Lugol's iodine instead of acetic acid. However, Schiller's test has poor specificity and was almost entirely replaced with the advent of cervical cytology.)

➢ **Treatment of Dysplasia and CIN**

1. CIN-I
   a. Treat the local infection
   b. Follow-up Pap tests every 3-6 months
   c. Persistent CIN-I requires treatment.
2. CIN-II and CIN-III
   a. Local destructive methods
      1) Cryosurgery
      2) Fulguration
      3) Electrocoagulation
      4) Laser ablation.
   b. Excision of abnormal tissue
      1) Cold knife conization
      2) Laser conization
      3) LLETZ.
   c. Surgery
      1) Therapeutic conization
      2) Hysterectomy
      3) Hysterectomy with removal of vaginal cuff.

➢ **Treatment of Invasive Carcinoma**

1. Stage IA1 – Cone or simple hysterectomy
2. Stage IA2, IB, IIA – Radical hysterectomy with bilateral pelvic lymphadenectomy, radiotherapy or combined therapy

3. Stage IIB, III, IV – Radiotherapy or combination with chemotherapy
4. Recurrent or persistent disease – Radical hysterectomy, pelvic exenteration operation, radiotherapy, chemotherapy for palliation, combined therapy.

Surgery for Ca cervix – Suitable patients include:

1. Young patients < 40 yrs age
2. FIGO stage IA to IIA
3. Associated conditions like fibroids, ovarian tumors, prolapsed uterus
4. Patients not willing for radiotherapy.

The surgical procedure includes radical hysterectomy with pelvic lymphadenectomy (Wertheim's hysterectomy). The uterus, cervix, surrounding parametrial tissues, cardinal ligaments, and upper third of vagina are removed. Lymph node groups which are removed include external and internal iliac, obturator and common iliac group. In young women the ovaries are not removed and the ovarian function is preserved.

Radiotherapy for Ca cervix – suitable patients include

1. FIGO stage IIB and IIIB
2. Older patients
3. Associated medical illnesses
4. Patient not willing for surgery.

Principles of radiotherapy: High dose gamma ray irradiation from intra-cavitary sources is given along with external RT with high energy megavoltage proton beams to the midplane of the pelvis, including the uterus, broad ligament and lateral pelvic wall.

Advantages of radiotherapy

- Less primary mortality and morbidity
- Successful in all stages of cancer
- Possible in those unfit for anesthesia
- Less risk of blood loss and transfusion.

Drawbacks of radiotherapy

- Narrowing of the vagina
- Proctosigmoiditis resulting in rectal ulcers and strictures
- Intestinal injuries
- Radiation cystitis
- Ureteric fibrosis and obstruction.

Post-op RT is indicated in patients with large tumors and those with positive pelvic lymph nodes.

Chemotherapy is used along with radiation as a radiosensitizer. Chemotherapeutic agents, e.g., Cisplatin, Bleomycin and Methotrexate are given to cause tumour regression before RT.

Prognosis:

5-year survival – 90% for stage I
70% for stage II
40% for stage III
5% – 10% for stage IV.

ENDOMETRIAL CARCINOMA

➢ **Risk factors**

1. Nulliparity
2. Unopposed estrogens
3. Endogenous – PCOD, anovulation, obesity
4. Exogenous – estrogen in HRT without progesterone(and presence of a uterus)
5. Late menopause
6. Tamoxifen treatment for Ca breast
7. Association with obesity, diabetes mellitus and hypertension called the 'corpus cancer syndrome'.

➢ **Types**

- Adenocarcinoma
- Adenosquamous carcinoma
- Papillary serous adenocarcinoma.

➢ **Symptoms**

1. Asymptomatic
2. Bleeding PV
3. Pain
   - Simpson's – referred in hypogastric and iliac fossa
   - Suprapubic – associated with pyometra
4. Symptoms of spread – headache, vomiting, dyspnea and hemoptysis.

➢ **Signs –** per abdomen – Ascites and hepatomegaly in advanced stage
– Palpable uterus (growth or hematometra)

➢ **Investigations**

Routine – complete blood count, RFT, LFT, chest X-ray, ECG

Specific

1. Endometrial aspiration and cytology
2. Fractional curettage – In this, endocervical curettage is followed by endometrial curettage with two samples examined separately. The fractional curettage determines the extent of spread of malignancy down the uterine wall so that the staging can be done and appropriate treatment planned
3. Hysteroscopy
4. D & C
5. USG, CT, MRI for staging
6. CA-125 increased in peritoneal involvement and extraperitoneal spread.

Staging

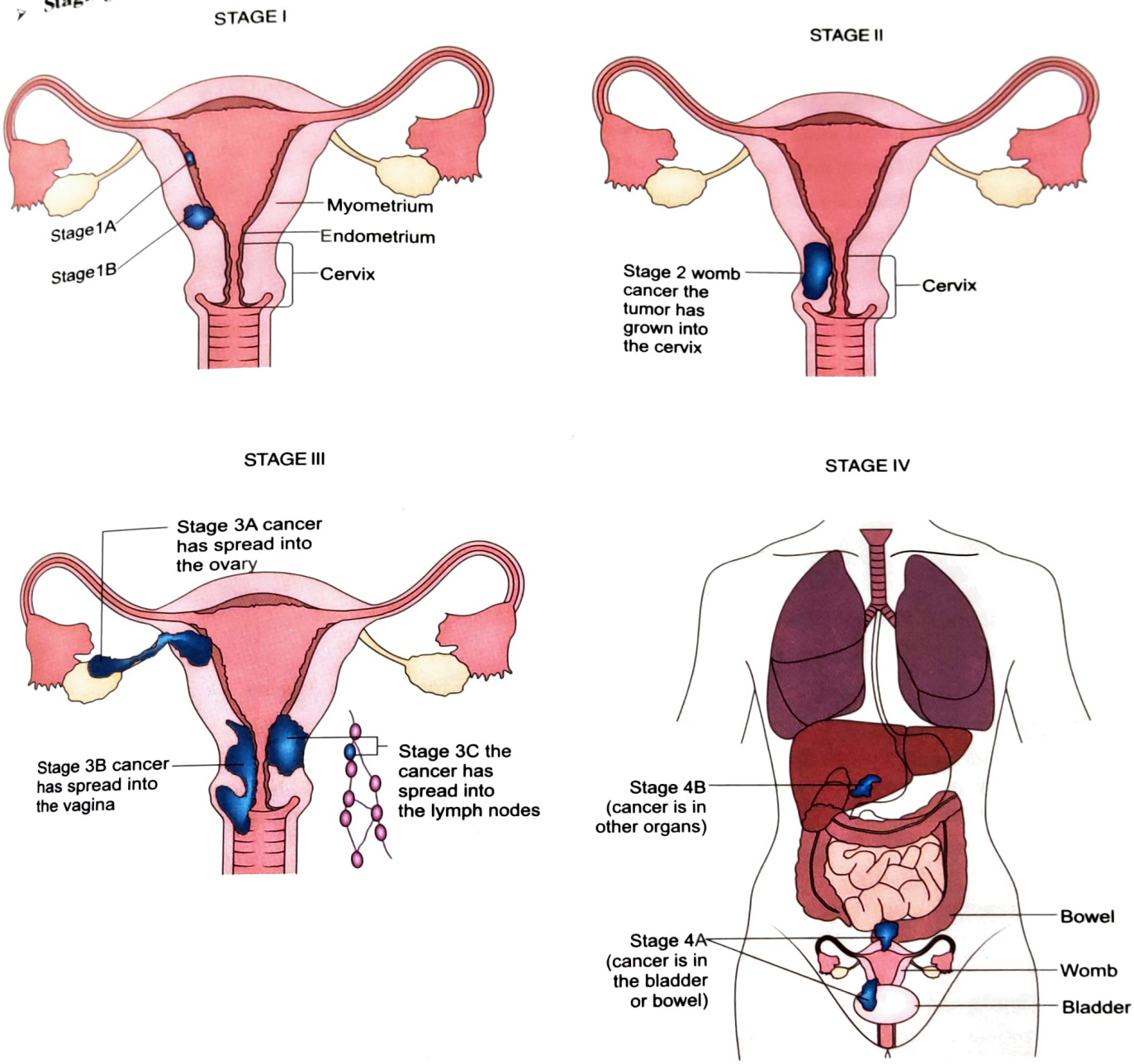


Fig. 25.4: Staging of endometrial carcinoma

Stage I	**Tumor confined to the corpus uteri**
IA	Myometrial invasion – none or < 50%
IB	Myometrial invasion – >50%.
Stage II	**Tumor invades cervical stroma, but does not extend beyond the uterus** (endocervical glandular involvement alone should be considered as stage I)
Stage III	**Local and/ or regional spread of the tumor**
IIIA	Tumor invades the serosa of the corpus uteri and/or adnexae
IIIB	Vaginal and/ or parametrial involvement
IIIC	Metastases to pelvic and/ or para-aortic lymph nodes
IIIC 1	Positive pelvic nodes
IIIC 2	Positive para-aortic lymph nodes with or without positive pelvic lymph nodes
Stage IV	**Tumor invades bladder and/or bowel mucosa, and/or distant metastases**
IVA	Tumor invasion of bladder and/or bowel mucosa
	Distant metastases, including intra-abdominal metastases with or without inguinal node metastases.

➢ **Treatment**

Stage I – Panhysterectomy (TAH+BSO)
Peritoneal wash
Omental biopsy
Node sampling

Stage II – Pelvic radiation with panhysterectomy (TAH+BSO)

Stage III & IV – Chemotherapy and radiotherapy

Follow-up every 3 monthly for first one year and 6 monthly for the next 3 years.

- ***S rger***

Total abdominal hysterectomy with bilateral salpingo oophorectomy. As part of surgical staging, peritoneal cytology, pelvic and para-aortic nodal sampling and omental biopsies should be done. Surgical staging provides prognostic information in order to justify the addition of adjuvant radiotherapy or chemotherapy.

- ***Radiotherap***

Adequate surgical staging will determine whether the patient should receive pelvic radiation, pelvic and para-aortic radiation or whole abdominal radiation. Primary treatment with radiation is used in patients who are not candidates for surgery. External beam radiation treatment to the pelvis is recommended for patients at high risk for local recurrence post surgery.

- ***Hormonal Therap***

Hormonal therapy, with progestational agents like medroxyprogesterone (Provera) and megestrol acetate, is used to treat advanced, recurrent or metastatic disease.

- ***hemotherap***

Patients with high-risk endometrial cancer (lymph node spread, papillary serous histology) are often offered adjuvant chemotherapy, sequentially with adjuvant radiotherapy. Chemotherapy is also used with palliative

intent for patients with recurrent or metastatic endometrial cancer. Combination therapy appears to be more effective than single agent therapy for advanced endometrial cancer. Carboplatin and paclitaxel is the most commonly used regime.

- **Prognosis**

5 year survival for Stage I carcinoma (all grades inclusive) is about 75%,
Stage II is 57%,
Stage III is 30% and
Stage IV is 10%.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. How will you screen a population for cancer cervix and write briefly the treatment options for cancer cervix stage I? 2. Enumerate the symptoms, signs and staging of endometrial carcinoma. Describe management 3. Describe staging of carcinoma cervix and suggest management option(s) for patient with stage III A disease. 4. Diagnosis and staging of carcinoma of cervix. Outline the treatment at various stages. 5. A 56 year old postmenopausal woman comes with the complaints of bleeding per vagina for 3 to 4 days for the past 3 months. She is obese and is a known diabetic on treatment. Discuss the investigations, diagnosis, differential diagnosis and management of this patient. 6. Discuss the signs, symptoms, diagnosis, differential diagnosis and management of malignant ovarian tumor. 7. Describe the diagnosis of Ca cervix and management of CIN. 8. Describe the diagnosis of Ca, complications and management of Ca cervix. 9. Describe the staging of Ca cervix. Discuss briefly management of stage Ib Ca cervix. 10. Describe staging of carcinoma cervix and suggest management option (s) for patient with Stage III A disease. 11. Discuss the Ca in situ of the cervix and its management. 12. How will you screen a population for cancer cervix and write briefly the treatment options for cancer cervix Stage III A. 13. What are the stages of cervical cancer? How would you manage Ca cervix IIa? 14. Discuss in detail the management cervical intraepithelial neoplasia.
Short Essays	1. Bartholin cyst diagnosis and management. 2. Gartner's cyst. 3. Colposcopy in cervical cancer screening. 4. Staging of carcinoma cervix. 5. FIGO classification of cancer cervix. 6. High risk factors for endometrial cancer. 7. Staging of carcinoma uterus and its risk factors.

Short Essays	8. Classify germ cell tumours of ovary and indicate tumor markers. 9. Features of malignancy in ovarian tumors on clinical examination and at laprotomy / scopy. 10. Write in brief "cancer screening" for cervical cancer. 11. Fractional curettage. 12. CIN cervix. 13. Radiotherapy of cervical cancer. 14. FIGO classification of cancer cervix. 15. Staging of carcinoma cervix. 16. Ca endometrium. 17. How will you do fractional curettage? 18. Describe the diagnosis and management of endometrial carcinoma. 19. Treatment of cervical intraepithelial neoplasia. 20. What is cervical-intraepithelial neoplasia? What is the treatment for CIN II? 21. Dysplasia of cervix. 22. Screening for cancer of cervix. 23. Microinvasive carcinoma of cervix. 24. Structures removed in Wertheim's hysterectomy.
Short Answers	1. Bartholin's cyst clinical features, complications and management 2. What are the risk factors for cervical cancer? 3. What is downstaging of cervical cancer? 4. Characteristics of Krukenberg tumor. 5. Schiller's test and its interpretation. 6. What is Mieg's syndrome? 7. Diagnosis of endometrial cancer. 8. Causes of CIN III. 9. List the signs and symptoms of a patient suffering from cancer cervix. 10. Epidemiological causes of carcinoma cervix. 11. Bartholin's abscess – clinical features and treatment. 12. Follicular cysts in a young lady. 13. Management of erosion of cervix. 14. Complications of cancer cervix. 15. Complications of radiotherapy. 16. Bartholin's abscess – clinical features and treatment. 17. Staging of cancer cervix. 18. Epidemiological factors for cancer of cervix. 19. Staging of endometrial carcinoma. 20. Treatment of stage IV cancer cervix. 21. Cold knife conization. 22. What are the predisposing factors for cancer endometrium? 23. What is erosion of cervix and how do you manage? 24. What is corpus cancer syndrome?

25. Fractional curettage.
26. HPV virus.
27. Indications for cone biopsy.
28. Treatment of cervical intraepithelial neoplasia III (CIN).
29. Survival rates in different stages of cancer of cervix.
30. Complications of radiotherapy.
31. Mention the groups of chemotherapeutic agents used in gynecological cancers.

Pelvic Inflammatory Disease

CHAPTER 26

PELVIC INFLAMMATORY DISEASE (PID)

- **Definition –** Inflammation of the upper genital tract involving the fallopian tubes, ovaries and surrounding structures.
- **Etiology**
  1. STD's – It is usually a polymicrobial infection. Gonococcal and chlamydial infections are most common. Other organisms include Mycoplasma, Tubercular bacillus, E. coli, Streptococcus, Staphylococcus, Bacteroides group, etc
  2. Delivery by dais
  3. Criminal abortions
  4. Minor operative procedures – D&C and hysterosalpingography
  5. Manual removal of placenta
  6. Evacuation of products of conception
  7. IUCD usage.
- **Clinical types**

  Acute PID

  Chronic PID – results from relapse of acute PID.
- **Clinical Features**
  1. Symptoms
     a. Bilateral lower abdominal and pelvic pain - dull in nature; onset is more acute in gonococcal (3 days) than in chlamydial (5-7 days) infection
     b. Vaginal bleeding – irregular and excessive
     c. Vaginal discharge – purulent and/ or copious
     d. Fever, lassitude and headache of varying intensity
     e. Nausea and vomiting
     f. Dyspareunia – painful sexual intercourse.
  2. Signs
     a. Temperature is elevated to beyond 38 C
     b. Abdominal palpation – tenderness on both the quadrants of lower abdomen; tender hepatomegaly may be present

c. Vaginal examination reveals:

1) Abnormal vaginal discharge to the extent of purulent

2) Congested external urethral meatus or openings of Bartholin's ducts through which pus may be seen escaping out on pressure

3) Speculum examination shows congested cervix with purulent discharge from the canal

4) Bimanual examination reveals bilateral tenderness on fornix palpation, which increase more with movement of the cervix. There may be thickening or a definite mass felt through the fornices.

Criteria for diagnosis of PID

Minim m criteria

- Lower abdominal tenderness
- Bilateral adnexal tenderness
- Cervical motion tenderness.

dditional criteria

- Oral temperature of >38 C
- Abnormal cervical or vaginal discharge
- Elevated ESR and C-reactive protein
- Laboratory documentation of cervical infection with Neisseria or Chlamydia.

Definiti e criteria

- Histopathological evidence of endometritis on endometrial biopsy
- Tubo-ovarian abscess on sonography or other radiologic tests
- Laparoscopic abnormalities consistent with PID.

omplications

1. Immediate:
   a. Pelvic peritonitis
   b. Generalized peritonitis
   c. Septicemia – producing arthritis or myocarditis
   d. Pelvic abscess.
2. Late:
   a. Dyspareunia
   b. Infertility – due to tubal damage or tubo-ovarian mass
   c. Chronic pelvic pain and ill health
   d. Increased risk of ectopic pregnancy

e. Chronic PID – due to recurrent or associated pyogenic infection; results in formation of adhesions, hydrosalpinx, pyosalpinx or tubo-ovarian mass.

A hydrosalpinx is a distally blocked fallopian tube filled with serous or clear fluid. The blocked tube may become substantially distended giving the tube a characteristic sausage-like or retort-like shape. The major cause for distal tubal occlusion is PID. A fallopian tube filled with pus is a pyosalpinx. Patients with hydrosalpinx resulting in infertility undergo IVF for conception.

➢ **Investigations**

1. Blood – hemoglobin, WBC count, ESR, culture, urea, serum electrolytes, CRP
2. Cervical and high vaginal swab culture
3. Culdocentesis – to rule out an ectopic pregnancy and to establish the diagnosis of a pelvic abscess
4. Laparoscopy – Findings in support of PID are:
   a. Hyperemia and edema of the tubes
   b. Exudates from the fimbrial ends
   c. Violin string - like adhesions in the pelvis and around the liver suggest chlamydial infection
   d. Pyosalpinx
   e. Pus in the pouch of Douglas
5. Ultrasound, CT, MRI – Dilated and fluid filled tubes, fluid in the pouch of Douglas or adnexal mass are suggestive of PID.

➢ **Treatment**

1. Medical Therapy
   a. Rest
   b. IV fluids – for dehydration, vomiting, electrolyte imbalance
   c. Analgesics – after confirmation of diagnosis
   d. Antibiotics – The effective antibiotics are:
      1) Erythromycin 500 mg qid for 7 days
      2) Clindamycin 450 mg qid for 10 days
      3) Tetracycline 500 mg qid for 10 days
      4) Doxycycline 100 mg bd for 10 days.
2. Surgical Therapy
   a. Indications
      1) Generalized peritonitis
      2) Pelvic abscess
      3) Tubo-ovarian abscess which does not respond to antimicrobial therapy.
   b. Methods
      1) Minimal invasive surgery in tubo-ovarian abscess – laparoscopic drainage, ultrasound-guided aspiration, percutaneous abscess drainage
      2) Conservative – salpingectomy, salpingo-oophorectomy
      3) Radical – abdominal hysterectomy with bilateral salpingo-oophorectomy.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. Mention common causes of PID. What are the clinical features? How do you treat chronic PID? 2. Describe acute salpingitis etiology, classification and management. 3. Discuss the clinical features, diagnosis and management of PID. 4. Define and describe the clinical features, risk factors and complications of pelvic inflammatory disease.
Short Essays	1. PID. 2. Definition and management of pelvic inflammatory disease. 3. Pelvic abscess. 4. Hydrosalpinx. 5. Differential diagnosis of acute pelvic inflammatory disease 6. Definition and management of pelvic inflammatory disease. Differential diagnosis of acute pelvic inflammatory disease. 7. Etiology and pathology of acute salpingitis. 8. Differential diagnosis of 24 weeks pelvic mass in 15 years girl. 9. Management of acute pelvic inflammatory disease (PID).
Short Answers	1. Definition of pelvic inflammatory disease and principles of management. 2. Causes of pyosalpinx. 3. Acute pelvic inflammatory disease write the symptoms.

CHAPTER 27

Chronic Pelvic Pain

- **Definition** – Chronic pelvic pain has been defined as pain of greater than 6 months in duration, localized to the anatomic pelvis and severe enough to cause functional disability or necessitating medical care.
- **Differential Diagnosis of Chronic Pelvic Pain**

Gynecologic	Non - Gynecologic
Non-cyclic • Adhesions • Endometriosis • Salpingo-oophoritis • Ovarian neoplasm benign or malignant • Uterine prolapse **Cyclic** • Primary dysmenorrhea • Mittelschmerz • Secondary dysmenorrhea • Endometriosis • Uterine or vaginal anomalies with obstruction of menstrual outflow • Intrauterine synechiae (Asherman's syndrome) • Endometrial polyps or intrauterine device (IUD) • Uterine fibroid • Adenomyosis	Irritable bowel syndrome Ulcerative colitis Infection Diverticulitis Interstitial cystitis Nerve entrapment syndrome Low back pain syndrome – Congenital anomalies – Scoliosis and kyphosis – Spondylolysis – Spondylolisthesis – Spinal injuries – Inflammation – Tumors

PREVIOUS EXAMINATION QUESTIONS

Long Essays	
Short Essays	1. Gynecological causes of low backache.
Short Answers	1. Causes of chronic pelvic pain.

Disorders of Broad Ligament, Fallopian Tubes and Parametrium

CHAPTER 28

PARAOVARIAN CYSTS

- **Definition** - These are extraperitoneal cysts lying in the broad ligament adjacent to the ovary, below the fallopian tubes.
- **Pathology**
  - The cyst is usually unilocular, contains clear fluid.
  - The cyst wall is smooth, thin and translucent.
  - Sometimes, a few loculi are present and papilloma may be scattered over the inner surface of the cyst.
  - The wall of the paraovarian cyst frequently contains smooth muscle.
- **Diagnosis**
  - Paraovarian cysts are clinically diagnosed as an ovarian cyst and at laparotomy it is identified as a broad ligament cyst.
  - Correct diagnosis is establised by the presence of muscle in the cyst on histological examination.
- **Treatment**

  Surgical removal when it attains large size.
- Para ovarian cysts types – Mullerian cyst, hydatid cyst of Morgagni, paratubal cyst.

PREVIOUS EXAMINATION QUESTIONS

Long Essays	
Short Essays	1. Paraovarian cyst.
Short Answers	1. Name the paraovarian cysts.

Endometriosis and Adenomyosis

CHAPTER 29

ENDOMETRIOSIS

- **Definition** – Occurrence of ectopic endometrial tissue outside the cavity of the uterus.
- **Sites**

1. Abdominal – It can occur at any site but is usually confined to the abdominal structures below the level of umbilicus. Common structures involved are:

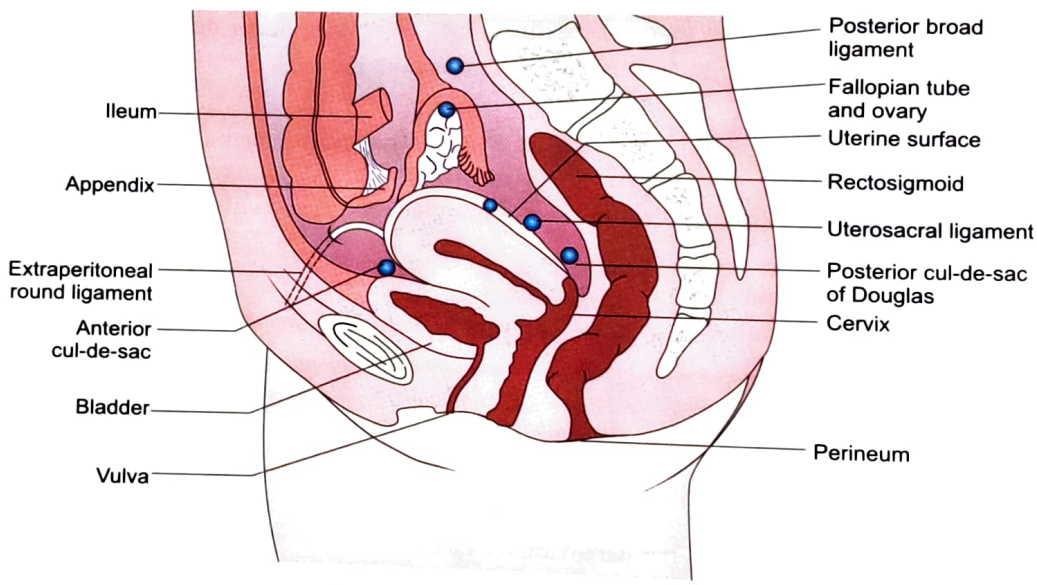


Fig. 29.1: Sites of Endometriosis

- Ovaries (A chocolate cyst is formed when a tiny patch of endometrial tissue bleeds, sloughs off, becomes transplanted, and grows and enlarges inside the ovaries. As the blood builds up over months and years, it turns brown)
- Pouch of Douglas
- Sigmoid colon
- Uterosacral ligament
- Back of uterus
- Broad ligament
- Intestinal coils

2. Extra-abdominal – The common sites are abdominal scar of hysterectomy, Caesarean section, tubectomy, myomectomy, umbilicus, episiotomy scar, vagina and cervix

3. Remote – pleura, lungs, deep tissues of arms and thighs.

Etiology

1. Implantation theory – reflux of menstrual endometrium through fallopian tube
2. Coelemic metaplasia theory – metaplasia of coelomic epithelium
3. Metastatic theory – embolisation of menstrual fragments through vascular or lymphatic channels
4. Histogenesis by induction – changes induced by chemical protein in endometrial cells called adhesion cytokine
5. Hormonal influence
6. Immunological factor – due to impaired T-cell and NK-cell activity and altered immunology.

Clinical Features

1. Symptoms
   a. About 30% of patients are asymptomatic
   b. Classic symptom complex includes
      Dysmenorrhea – premenstrual pain which is releaved with menses
      Dyspareunia – due to involvement of POD and uterosacral ligament causing adhesion of uterus
      Menorrhagia – mainly seen in adenomyosis
      Infertility – due to interference in tubal motility and function and reduced coitus due to dyspareunia
   c. Chronic pelvic pain – due to PG E_2 in the peritoneal fluid
   d. Abdominal pain – in case of ruptured chocolate cyst
   e. Urinary disturbances – frequency, dysuria, hematuria
   f. Bowel disturbances – painful defecation, diarrhea, melaena.
2. Signs
   a. Abdominal examination
      1) May not reveal any abnormality
      2) Mass may be felt in the lower abdomen arising from the pelvis – enlarged chocolate cyst or tubo-ovarian mass due to endometriotic adhesions
      3) Mass is tender with restricted mobility.
   b. Bimanual examination
      1) May not reveal any pathology
      2) Positive findings are pelvic tenderness, nodules in the pouch of Douglas, nodular feel of the uterosacral ligaments, fixed retroverted uterus or unilateral or bilateral adnexal mass of varying sizes
   c. Speculum examination – bluish spots in the posterior fornix.

Classification

Stage I – Minimal – Small spots seen on laparoscopy. No symptoms.
Stage II – Mild – Scattered superficial lesions. No adhesions.
Stage III – Moderate – Ovaries involved (<2 cm) with scarring.
Stage IV – Severe – Ovaries with endometrioma (>2 cm) with thickened uterosacral ligaments along with involvement of bowel and bladder.

Complications

1. Endocrinopathy – anovulation, abnormal follicular development, hyperprolactinemia

2. Rupture of chocolate cyst
3. Infection of chocolate cyst.

➢ **Investigations**

1. Laparoscopy – The classic lesion of pelvic endometriosis is described as 'powder burns' seen on the uterosacral ligaments or 'match stick' spots on the peritoneum of the pouch of Douglas. Scarring in the peritoneum surrounding the implants is also a typical finding.
2. Serum marker CA-125 – raised to more than 35 U/ ml and the level is directly proportional to the extent of the disease.
3. Ultrasound, CT, MRI – not very helpful in the diagnosis.
4. Biopsy – for doubtful or excised lesion.

➢ **Treatment**

1. Asymptomatic minimal cases
   a. Observe 6-8 months
   b. Investigate infertility.
2. Symptomatic cases
   a. Drug treatment
      1) OCPs
      2) Progestogens
      3) Androgens
      4) GnRH analogues.
   b. Minimal invasive surgery – laparoscopy with:
      1) Destruction by cautery or laser vapourization
      2) Excision of cyst
      3) Presacral neurectomy
      4) LUNA (laparoscopic uterosacral nerve ablation) for pain.
   c. Surgery – laparotomy with:
      1) Incision of chocolate cyst and removal of lining
      2) Salpingo-oophorectomy
      3) Hysterectomy with bilateral salpingo-oophorectomy
      4) Excision of scar endometriosis.

ADENOMYOSIS

➢ **Definition** – condition where there is in-growth of the endometrium, both the glandular and stromal components, directly into the myometrium.

➢ **Etiology** – not known; may be related to:

1. Repeated childbirths
2. Vigorous curettage
3. Estrogen excess which produces cystic glandular hyperplasia of the endometrium.

➢ **Clinical Features**

1. Symptoms
   a. Asymptomatic in one-third of cases
   b. Patients are usually parous and above 40 years of age
   c. Menorrhagia
   d. Dysmenorrhea
   e. Dyspareunia
   f. Pelvic discomfort
   g. Backache
   h. Frequency of micturition.
2. Signs
   a. Abdominal Examination – hypogastric mass arising out of the pelvis and occupying the midline; symmetrically enlarged to not more than 14 weeks pregnant uterus size

b. Pelvic examination – uniform enlargement of the uterus.

- **Investigations** – Ultrasound and Color Doppler (TVS) reveal:
  1. Heterogenous echogenecity
  2. Hypoechoic myometrium
  3. Multiple small cysts in the myometrium (bleeding)
  4. Increased vascularity within the myometrium.
- **Treatment** – The treatment is surgical. There is no place of hormone therapy.
  1. Conservative – partial resection
  2. Radical – total hysterectomy with or without salpingo-oophorectomy

PREVIOUS EXAMINATION QUESTIONS

Long Essays	1. Enumerate causes of congestive dysmenorrhea. What are the treatment options for pelvic endometriosis? 2. Theories, clinical features and management of endometriosis. 3. Define endometriosis. Describe clinical features and management. 4. Clinical features, diagnosis and treatment of adenomyosis at 35 years. 5. Describe the endometriosis. Describe the etiology, diagnosis and management 6. Describe the endometriosis. Describe the etiology, diagnosis and management of pelvic tuberculosis. 7. Describe the clinical features, diagnosis and treatment of pelvic endometriosis. 8. Discuss the etiology of endometriosis and diagnosis and its medical management.
Short Essays	1. Theories of endometriosis. 2. Chocolate cyst of ovary. 3. Diagnosis and management of chocolate cyst of ovary. 4. Medical treatment of endometriosis. 5. Surgical management of ovarian endometriosis. 6. Adenomyosis–diagnosis and management. 7. Diagnosis and management of adenomyosis. 8. Endometriosis–symptoms and signs. 9. Medical management of endometriosis. 10. Pathogenesis of endometriosis. 11. What is ovarian endometrioma? What is the medical management of endometriosis? 12. Treatment of endometriosis. 13. Medical treatment of endometriosis.
Short Answers	1. Causes of symmetrical enlargement of uterus. 2. Medical management of endometriosis. 3. Theories of endometriosis. 4. What is adenomyosis? What are the presenting symptoms? 5. Danazol in endometriosis. 6. Adenomyosis.

Aids in Gynecology

CHAPTER 30

LAPAROSCOPY

➢ **Indications**

1. Diagnostic
   a. Infertility work up
   b. Chronic pelvic pain
   c. Pelvic mass – fibroid, ovarian cyst
   d. Acute pelvic lesion – ectopic, acute appendicitis, acute salpingitis
   e. Follow- up of pelvic surgery – tuboplasty, ovarian malignancy
   f. Investigation protocol of amenorrhea
   g. Diagnosis of suspected Mullerian abnormalities
   h. Uterine perforation.
2. Therapeutic
   a. Minor Procedures
      1) Tubal sterilization
      2) Adhesiolysis (without bowel involvement)
      3) Aspiration of simple ovarian cysts
      4) Ovarian biopsy.
   b. Moderate Procedures
      1) Ectopic
         a) Salpingostomy
         b) Salpingectomy
         c) Salpingo-oophorectomy
         d) Segmental resection
      2) Endometriosis – ablation by diathermy or laser

3) Ovary
   a) Diathermy for PCOD
   b) Drainage of endometriomas
   c) Ovarian cystectomy
4) Uterus
   a) Laparoscopic assisted vaginal hysterectomy
   b) Adhesiolysis (with bowel involvement).

c. Extensive procedures – Surgery for:
   1) Major endometriosis
   2) Myomectomy
   3) Pelvic lymphadenectomy
   4) Urinary incontinence.

➢ **Contraindications**

1. Severe cardiopulmonary disease
2. Patient hemodynamically unstable
3. Significant hemoperitoneum
4. Generalized peritonitis
5. Extensive peritoneal adhesion
6. Intestinal obstruction
7. Large pelvic tumor
8. Pregnancy >16 weeks
9. Previous periumbilical surgery
10. Extreme obesity.

➢ **Complications**

1. Specific to laparoscopy
   a. Extra-peritoneal insufflation – surgical emphysema
   b. Omental emphysema
   c. Cardiac arrhythmia
   d. Injury to blood vessels
   e. Injury to organs – bowel, bladder or ureter
   f. Electro-surgical complications – causing thermal injury
   g. Gas (carbon dioxide) embolism.
2. Due to anesthesia
   a. Hypoventilation
   b. Hypercarbia and metabolic acidosis
   c. Basal lung atelectasis
   d. Esophageal intubation
   e. Aspiration
   f. Cardiac arrest.

3. Common to any surgical procedures
   a. Infection
   b. Hemorrhage
   c. Wound dehiscence
   d. Incisional hernia.
4. The potentially fatal complications associated with laparoscopy are:
   a. Cardiopulmonary arrest
   b. Hemorrhage
   c. Gas embolism
   d. Cautery burns.

HYSTEROSCOPY

➢ **Indications**

1. Abnormal uterine bleeding
   - Ablation of endometrium (pre-treatment with GnRH agonist)
   - Excision of endometrial polyps, submucous fibroids
   - Directed endometrial biopsy.
2. Infertility
   - Division of endometrial adhesions (e.g. Asherman's syndrome)
   - Uterine septum resection.

➢ **Complications**

1. Acute and chronic upper genital tract infections
2. Profuse bleeding
3. Cervical stenosis
4. Recent uterine perforation.

ULTRASOUND IN GYNECOLOGY

➢ 3.5 MHz frequency is used in abdominal ultrasound where as 5-7.5 MHz is used in vaginal type.
➢ Higher is the frequency more will be the resolution of the image but lower will be the depth of tissue penetration.

➢ **Types**

1. Transabdominal
2. Transvaginal.

➢ **Indication**

1. Infertility – for folliculometry and endometrial thickness
2. Diagnose ovulation, time of ovulation
3. Ectopic pregnancy
4. Oocyte retrieval in IVF
5. Ovarian masses like cysts and tumors
6. Uterine fibroid, tubo-ovarian masses

7. Oncology – color Doppler – low impedance and high flow velocity suspects of malignancy
8. Endometrial diseases – polyps, fibroid, growths
9. Misplaced IUCD
10. DUB or AUB and post menopausal bleeding
11. Pelvic pain causes
12. Molar pregnancy
13. Malformations of uterus and vagina.

SALINE SONOHYSTEROGRAM

Saline infusion sonohysterography (SHG or SIS) consists of imaging the uterus and uterine cavity using ultrasonography while sterile saline is instilled into the uterine cavity. The purpose of sonohysterography is to detect abnormalities of the uterus and endometrial (uterine lining) cavity.

Indications

1. Abnormal uterine bleeding
2. Infertility
3. Recurrent spontaneous miscarriage
4. Congenital abnormalities of the uterus
5. Preoperative and postoperative evaluation of the uterine cavity
6. Suspected intrauterine adhesions (synechiae or scar tissue).

Contraindications

1. Pregnancy
2. Acute pelvic infection.

Procedure

SHG is usually performed in the first part of the menstrual cycle after menstruation. The procedure begins with a transvaginal ultrasound examination. After this is completed, a narrow catheter is placed vaginally through the cervix into the uterine cavity, and the ultrasound examination is continued while sterile saline is instilled into the uterine cavity. The saline solution helps outline the uterine cavity to show abnormalities, such as endometrial polyps and intracavitary fibroids.

PREVIOUS EXAMINATION QUESTIONS

Short Essays	1. Laparoscopy. 2. Diagnostic laparoscopy complications. 3. Indications of endoscopic surgeries in gynecology. 4. Colposcopy – principle and indications. 5. Colposcopy in cervical screening. 6. Hysteroscopy. 7. Uses or place of laparoscopy in gynecology. 8. Role of diagnostic laparoscopy. 9. What is saline sonohysterogram?

	10. Indications for ultrasound in gynecology. 11. Ten uses of ultrasound in gynecology. 12. Indications of transvaginal sonography in gynecology.
Short Answers	1. Mention 6 indications that can be diagnosed by laparoscopy. 2. Preparations of per vagina and steps of laparoscopic tubal ligations. 3. Enumerate the indications and complications of diagnostic laparoscopy. 4. Indications for hysteroscopy. 5. Contraindications for laparoscopic surgery. 6. Use/ role of ultrasound in gynecology. 7. Indications for ultrasound in gynecology.

Operative Gynecology

CHAPTER 31

HYSTERECTOMY

Types of Hysterectomy

Depending on the extent of removal of the uterus and the adjacent structures, the following types of hysterectomy are described:

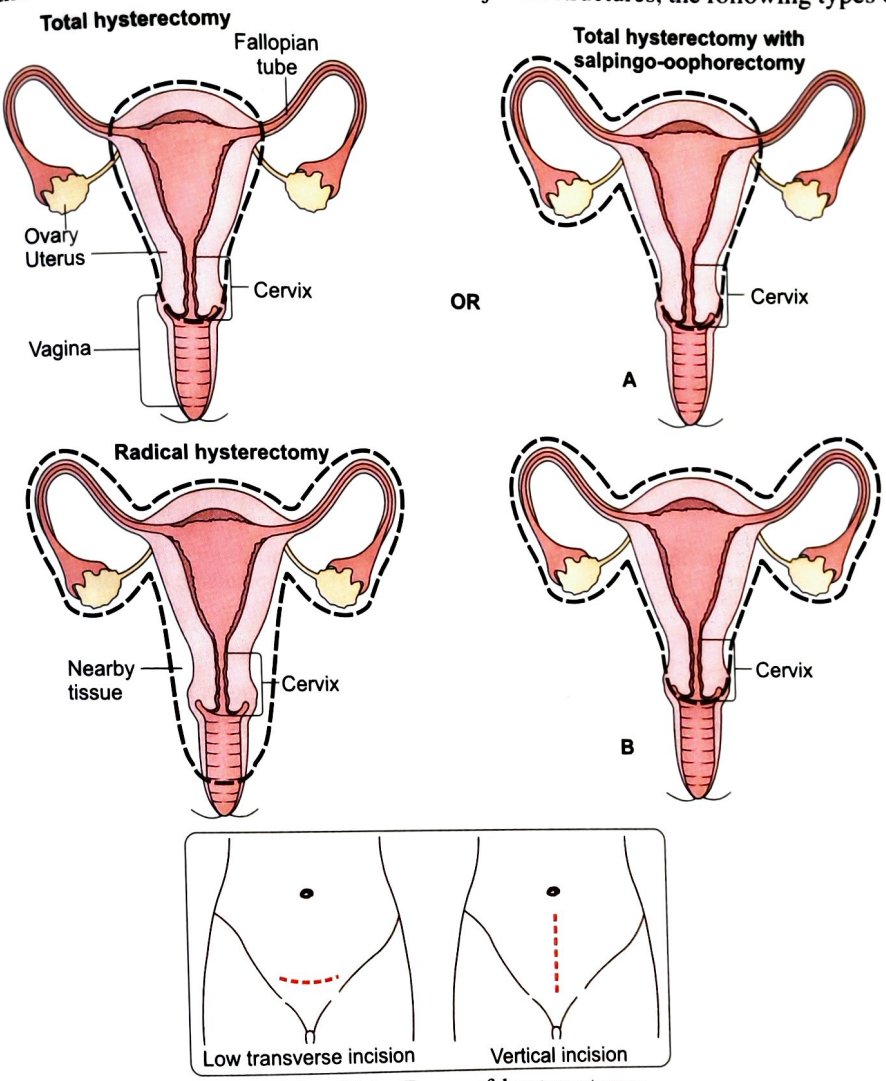


Fig. 31.1: Types of hysterectomy

a. Total – removal of the entire uterus
b. Subtotal or supracervical – removal of only the body or corpus of the uterus leaving the cervix behind
c. Hysterectomy with bilateral salpingo-oophorectomy – earlier known as 'panhysterectomy', involves removal of the uterus with both fallopian tubes and ovaries
d. Extended hysterectomy – removal of uterus, both fallopian tubes and ovaries with a cuff of vagina
e. Radical hysterectomy – removal of uterus, both fallopian tubes and ovaries, upper one third of vagina, adjacent parametrium and pelvic nodes.

➢ **Indications**

Benign lesions

a. Fibroid uterus
b. Dysfunctional uterine bleeding unresponsive to medical treatment
c. Uterovaginal prolapse
d. Endometriosis, adenomyosis
e. Chronic pelvic pain
f. Chronic pelvic inflammatory disease with persistent symptoms
g. Cervical intraepithelial neoplasia (CIN) of high grade in a woman who has completed her family and cannot come for follow-up
h. Complex endometrial hyperplasia.

Malignant lesions

a. Carcinoma cervix
b. Carcinoma endometrium
c. Carcinoma ovary and fallopian tube
d. Uterine sarcoma
e. Choriocarcinoma in rare specified circumstances.

Obstetric

a. Massive atonic postpartum hemorrhage unresponsive to medical and other surgical interventions.
b. Morbidly adherent placenta–placenta accreta/ increta/ percreta
c. Septic abortion (selected cases)
d. Cervical ectopic pregnancy
e. Traumatic as a last resort for uncontrolled haemorrhage when dealing with
   * Extensive cervical tears which cannot be repaired vaginal
   * Large uterine perforation in which uterus cannot be repaired
   * Rupture uterus (in a parous woman)
   * Incorrectable uterine inversion.

➢ **Routes of performing a hysterectomy**

1. Abdominal
2. Total vaginal
3. Laparoscopic assisted vaginal
4. Total laparoscopic.

The vaginal route is the least traumatic and utilizes a "natural access" i.e., the vagina. It is much like a vaginal delivery with rapid patient recovery.

The laparoscopic approach is a "minimal access" approach since only small cuts/ punctures in the abdomen are used to perform the procedure.

In abdominal approach, a transverse muscle splitting (Pfanensteil) or muscle cutting incision (Maylard) is used. In patients with large uteri, multiple fibroids or malignancy a midline or paramedian vertical abdominal incision is used. Abdominal hysterectomy has a higher incidence of complications, a longer length of hospital stay and convalescence.

Counselling and consenting

An informed consent must be obtained prior to performing a hysterectomy. The discussion should include:

- Issues pertaining to surgery, nature and extent of the disease process, extent of the proposed surgery, the potential modifications of the operation depending on the intraoperative findings, the anticipated benefits of the operation, and the potential complications of surgery.
- Alternative methods of therapy available, the risks and results of the same.
- The likely outcome if the patient is not treated at all.
- The need for hormone replacement therapy in the postoperative period after a bilateral oophorectomy along with its risks and benefits.
- Pre-anesthetic evaluation.

Steps of the operation

In an abdominal hysterectomy, the steps of performing a hysterectomy after the abdomen has been opened are detaching the uterus from its attachments in a craniocaudal sequence as given below:

i. Detachment from round ligaments
ii. Detachment of the utero-ovarian or infundibulopelvic ligaments after identification of the ureter crossing the common iliac artery
iii. Mobilization of the urinary bladder after detaching the uterus from the uterovesical fold of peritoneum
iv. Separation of anterior and posterior leaves of the broad ligament
v. Ligation and detachment of uterine vessels at the junction of cervix and the body of the uterus
vi. Detachment after incision of the posterior peritoneum to free the uterus from its rectovaginal fold of peritoneum (pouch of douglas)
vii. Detachment of cardinal ligaments
viii. Detachment of cervix from its vaginal wall attachments
ix. Removal of uterus and cervix (with a cuff of vagina in radical hysterectomy)
x. Closure of the vaginal vault
xi. Re-approximation of the pelvic peritoneum (optional)
xii. Closure of the abdomen.

In vaginal hysterectomy, the steps include:

i. Incising the vagina circumscribing the cervix
ii. Dissection of the vaginal wall off the cervix
iii. Opening the posterior cul-de-sac to detach the rectovaginal peritoneum
iv. Detachment of the uterus from its attachment to the uterosacral ligaments
v. Entry into vesicovaginal space to detach the vesicouterine fold of peritoneum

vi. Detachment of the uterus from the attachment of the cardinal ligaments
vii. Advancement of bladder with any adhesiolysis if bladder is adherent to the lower uterine segment
viii. Detachment of the uterine artery as it approaches the uterus
ix. Utero-ovarian and round ligament ligation
x. Ligation of infundibulopelvic ligaments if adnexa are removed
xi. Removal of the uterus and cervix
xii. Ensuring hemostasis
xiii. Peritoneal closure
xiv. Vaginal mucosal closure
xv. Anchoring the vault If vaginal hysterectomy is being performed for prolapse, the uterosacral ligaments are anchored to the vaginal vault to prevent subsequent vault prolapse. Pelvic floor repair is done concomitantly.

In a laparoscopic assisted vaginal hysterectomy (LAVH), the laparoscopic part of the operation begins with the mobilization of the uterus and adnexa from the pelvic side walls by its attachments to the round ligament and the infundibulopelvic ligament. The uterine vessels may be divided laparoscopically or vaginally. Usually the uterosacral and the cardinal ligaments are clamped and cut vaginally and the uterus and cervix delivered per vaginum.

In total laparoscopic hysterectomy, the entire procedure is performed laparoscopically including closure of vaginal vault.

➢ **Postoperative monitoring, care and recovery**

a. Monitoring of vital signs and vaginal bleeding
b. Provision of adequate analgesia
c. Provide bladder drainage and urinary catheterization
d. Ensure mobilization and activity
e. Ensure adequate calorie and fluid intake
f. Watch bowel movement for return of bowel activity
g. Continue thromboprophylaxis
h. Provide chest and leg physiotherapy
i. Assess requirement of transfusion of blood or blood products
j. Continue antibiotic cover in patients with infection.

➢ **Complications of hysterectomy**

Intraoperative complications

a. Hemorrhage
b. Urinary tract injuries
c. Gastrointestinal injuries
d. Other visceral injuries
e. Nerve injuries
f. Vascular injuries
g. Those related to the laparoscopic route and pneumoperitoneum

Immediate postoperative complications

a. Reactionary hemorrhage
b. Pyrexia
c. Acute pelvic infection and abscess
d. Thrombophlebitis
e. Paralytic ileus
f. Urinary tract infection
g. Lung complications from aspiration and infection
h. Deep vein thrombosis
i. Pulmonary embolism
j. Depression and psychological problems.

Long term complications

a. Intestinal obstruction
b. Urinary tract infection
c. Urogenital fistula
d. Fallopian tube prolapse
e. Persistence of chronic pelvic pain
f. Sexual dysfunction and depression.

DILATATION AND CURETTAGE +/- HYSTEROSCOPY

➢ **Procedure**

- Patient placed in dorsal lithotomy position
- Pelvic examination under anesthesia to confirm orientation and size of uterus
- Cervix exposed and grasped on anterior lip with single-toothed tenaculum
- Kevorkian curet used to scrape endocervical canal and obtain specimen
- Uterus is then sounded (measured); normal uterus size <8 cm along internal longitudinal axis
- Cervix then dilated sequentially to 9 mm
- Hysteroscope inserted at this point if hysteroscopy to be done; copious irrigation fluid (preferably glycine; also carbon dioxide, cytosol) is used to distend endometrial cavity; small biopsy forceps can be inserted into this port to sample tissue of interest under direct visualization
- After hysteroscopy completed, sharp curettage done by gently scraping all sides of uterus with curette
- All instruments removed and cervix inspected for bleeding.

➢ **Indications**

Diagnostic

- DUB
- Sterility
- Amenorrhea/oligomenorrhea
- Malignant disease of uterus.

Therapeutic

- Removal of retained products of conception following abortion
- Therapeutic termination of pregnancy

- Removal of polypi and small submucous fibroids
- Removal of IUD
- Drainage of pyometra/ hematometra.

Complications

- Bleeding
- Perforation
- Infection
- Absorption of excess distension medium

PREVIOUS EXAMINATION QUESTIONS	
Short Essays	1. Dilatation and curettage. 2. Technique of vaginal hysterectomy. 3. Preoperative preparations for hysterectomy. 4. Prevention of vault prolapse in vaginal hysterectomy.
Short Answers	1. Six indications for D & C. 2. Diagnostic dilatation and curettage. 3. Complications of vaginal hysterectomy. 4. Prevention of vault prolapse in vaginal hysterectomy. 5. Intraoperative complications of hysterectomy. 6. Daywise postoperative management after abdominal hysterectomy.